# A CLINICAL STUDY OF OCULAR MOTOR NERVE PALSIES

*This dissertation is submitted for*

**M.S. DEGREE EXAMINATION**

**BRANCH – III OPHTHALMOLOGY**

**MARCH 2010**

**THE TAMILNADU**

**DR. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI, TAMILNADU**

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# CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL STUDY OF OCULAR MOTOR NERVE PALSIES"** has been done by **Dr. S. SUMATHY,** under my guidance in Department of OPHTHALMOLOGY, Coimbatore Medical College Hospital, Coimbatore.

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# DECLARATION

I, **Dr. S. SUMATHY,** solemnly declare that the dissertation titled "**A CLINICAL STUDY OF OCULAR MOTOR NERVE PALSIES**" has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S., (Ophthalmology) Branch – III Degree Examination to be held in March 2010.

**Place : Coimbatore**

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# ACKNOWLEDGEMENT

I am grateful to the **Dean Dr. V. Kumaran, MS, MCh.,** Coimbatore Medical College Hospital, Coimbatore for permitting me to do this study.

I am extremely thankful to **Prof. V. R. Vijayaraghavan MS, DO, DNB, FRCS(Ed),** Head of the Department, Ophthalmology, Coimbatore Medical College Hospital for his valuable guidance in the preparation of this dissertation.

My sincere thanks are due to **Prof. A. Rajendra Prasad MS DO** for his valuable advice and encouragement which helped me in completing this work successfully.

It is my pleasant duty to express my gratitude and thanks to **Dr. K. Govindarajan MD, DM** professor of Neurology for his encouragement.

My thanks are also due to **Dr. Paramasivan DO, Dr. P. Sumathi MS, Dr. J. Saravanan MS and Dr. C. Jeevakala MS, DO** for helping and guiding me in completing this work.

Finally I would like to place my sincere thanks to all my patients for their immense cooperation with out which this study wound not have been possible.

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# A CLINICAL STUDY OF OCULAR MOTOR NERVE PALSIES

## INTRODUCTION

The designation "Ocular motor system" refers to the entire somatic motor system that controls the position and movements of the eyes. This system includes the extraocular muscles, the cranial nerves and nuclei that innervate them and the forces that stimulate and inhibit their actions. Restricted Ocular mobility could be the result of paralysis of the nerves supplying the extra ocular muscles (neurogenic) or due to the pathology in the muscle itself (myogenic) or there may be the pathology at the myoneural junction (neuro muscular).

Since the treatment for these conditions is different from one another, it becomes clinically important to differentiate them, thus avoiding elaborate neurological investigations it is not necessary.

Movements of the eye ball is brought about by six extraocular muscles – the four recti and the two obliques. The cranial nerves supplying the extra ocular muscles are - the Oculomotor nerve (third), the Trochlear nerve (fourth) and the Abducens nerve (sixth). The Abducens nerve supplies the lateral rectus muscle and the trochlear supplies the Superior oblique and the third nerve supplies the remaining muscles along with levator palpebrae superioris and the intrinsic muscles of the eye.

Palsy is the term used to denote either paresis (partial) or Paralysis (total) of that particular nerve. These nerve palsies could be either Congenital or acquired. Most congenital palsies occur as isolated defects in an otherwise healthy individual,

presumed to be due to developmental defects in the nucleus or the nerve pathway. Acquired palsies are more common and the etiology varies. Ocular motor system involvement can be divided anatomically and physiologically into infranuclear or peripheral (commonest) nuclear, internuclear (MLF) and supranuclear (brainstem, cerebral, vestibular and cerebellar) components.

## ANATOMY

## OCULOMOTOR NERVE OR THE III NERVE

The oculomotor nucleus is located in the periaqueductal gray matter inferior to the sylvian duct at the level of the superior colliculus. It consists of 2 main somatic lateral nuclei source of somatic fibres to the ocular muscles and the forked anterior group of small cells- Edinger-Westphal nucleus supplying parasympathetic innervation to the eye. A single caudal midline group of cells innervates both levator muscles. The medial rectus, inferior rectus and inferior oblique muscles receive their innervation from the appropriate ipsilateral subnucleus while superior rectus receives innervation from the contralateral superior rectus subnucleus.

The fascicular portion, starting from the ventral side of the nucleus runs through the parenchyma of the midbrain and pass laterally through the red nucleus to exit above the medial aspect of the cerebellar peduncles into the interpeduncular fossa.

The basilar portion of the nerve leaves the midbrain as 15,000 fibers at the sulcus oculomotorius between 2 cerebral peduncles and then coalesce to form the main trunk. It passes through the subarachnoid space, pierces the durometer lateral to the posterior clinoid process and enters the cavernous sinus. Within the subarachnoid space, the nerve passes between the superior cerebellar artery and the posterior

cerebellar artery and is located parallel and lateral to the posterior communicating artery.

The cavernous part in the lateral wall of the cavernous sinus superior to the other nerves and divides into the superior and inferior branch in the anterior part.

The nerve enters the orbit through the superior orbital fissure. The superior division innervates the levator and superior rectus muscles. The larger inferior division innervates the medial rectus, inferior rectus and inferior oblique muscles and parasympathetic fibres to sphincter pupillae and ciliary muscle through the ciliary and episcleral ganglion.

The location of the parasympathetic, pupillomotor axons within the third nerve is important. The pupillomotor fibres are peripheral and located in the superomedial part of the nerve as it leaves the brain stem. Within the cavernous sinus the pupillomotor fibres which are located centrally descend through the substance of the oculomotor nerve in the middle third of cavernous sinus. As the nerve enters the orbit, they are located in the inferior peripheral portion. The pupillomotor fibres are smaller and more thinly myelinated than the somatic fibres of the oculomotor nerve.

## THE FOURTH NERVE OR TROCHLEAR NERVE

The fourth nerve nucleus is located in the periaquaductal gray matter ventral to the sylvian aqueduct at the level of inferior colliculus, caudal to the oculomotor nucleus. Each nerve innervates contralateral superior oblique muscle. The fasciculus leaves the nucleus laterally and curves in a dorsal and caudal direction to decussate completely in the anterior medullary velum. The nerve leaves the brain stem just below the inferior colliculus on the dorsal surface.

The subarachnoid portion of the nerve (containing approximately 2000 fibers) curves forward around the brainstem, runs beneath the tentorial free edge and passes between the posterior cerebral and the superior cerebellar arteries. It passes in a rostral direction, pierces the dura and enters the cavernous sinus, lateral and inferior to the third nerve. In the anterior portion of the cavernous sinus the fourth nerve rises and enters the orbit through the superior orbital fissure above the annulus of Zinn to innervate the superior oblique muscle.

## ABDUCENS OR SIXTH CRANIAL NERVE

The nucleus of VI nerve (Abducens nerve), is located in the pons inferior to the floor of IV ventricle and slightly lateral to the midline anterior to the pontomedullary junction. Adjacent and medial to each nucleus is the PPRF. The MLF is located superior medial to the VI nerve nucleus the nucleus is closely related to the fasciculus of VII nerve – the facial colliculus. The nerve (approximately 4000 – 6000 fibers) emerges ventrally at the pontomedullary junction just lateral to the pyramidal prominence and enters the prepontine basilar cistern. The nerve which has the longest subarachnoid course passes upward along the base of pons on either side of basilar artery above the clivus. It lies adjacent to or passes through Dorello's canal formed by the petroclinoid ligament (Gruber's ligament) within the canal, VI nerve travels with the inferior petrosal sinus and enters the cavernous sinus by perforating the dura matter lateral and inferior to the III and IV nerves. It is the most medial cranial nerve in the cavernous sinus and is closest to the carotid artery. The nerve enters the orbit through the superior orbital fissure through the annulus of Zinn and terminates at the lateral rectus muscle.

## OCULAR MOTOR NERVE PALSIES

Oculomotor system can be anatomically and physiologically divided into

- Supranuclear component (brain stem, cerebral, ventricular and cerebellar lesions).
- Nuclear and internuclear lesions.
- Intranuclear component or peripheral component:

Commonest and is the bulk of the cases under study. Lesions of the infranuclear oculomotor system may affect the III, IV & VI nerve at any location from their fascicular portion to the termination in the extraocular muscles. The lesion may affect two or more of the nerve in combination in one or both eyes and may present as follows:

## TOTAL OPHTHALMOPLEGIA

It is the paralysis of all the EOM and LPs and the intraocular muscles (Ciliary muscle and pupillary muscle). This is characterized by complete ptosis, slight protopsis of the eyeball, in a position of slight divergence with a large dilated pupil and absence of accommodation.

### Causes

1. Wide spread inflammation or vascular lesions of brainstem – usually bilateral.
2. Extramedullary course, the cavernous sinus or superior orbital fissure or orbit – unilateral.

**External Ophthalmoplegia**

Paralysis of EOM & LPS where pupillary activity and accommodation are spared.

**Internal Ophthalmoplegia**

Palsy of only internal muscles (Ciliary and Pupillary muscles).

**Partial Ophthalmoplegia**

Palsy of EOM where one or more of the external muscles have escaped. Usually orbital causes and unilateral.

**Ocular Motor Palsies**

May present in one of the 4 ways.

1. Isolated partial or complete nerve palsies without any other neurologic signs and without other symptoms.
2. In associated with symptoms (Pain, dysesthesia, paraesthesia etc.) but without any signs of neurologic or systemic disease.
3. In association with other ocular motor nerve palsies but without any other neurologic signs.
4. In association with neurologic signs.

## CONGENITAL VS ACQUIRED PALSIES

### Importance

1. Cause must be found and treated in acquired palsies.
2. No further investigation is needed if congenital.
3. Immediate treatment in congenital, at least 6 months allowed for spontaneous recovery if acquired.

## CHARACTERISTICS OF MUSCLE PARALYSIS

Objective signs of nerve palsies are (i). Abnormal deviation of eyes. (ii). Compensatory head posture (iii) Limitation of Movements.

Subjective symptoms are (i). Diplopia (ii). False orientation (iii). Ocular vertigo.

1. Compensatory head posture

In general, any ocular motor nerve palsy will result in limitation of ocular movement leading to incomitant or paralytic strabismus causing diplopia. The direction of deviation depends on the nerve affected. Paralytic strabismus increases as the eyes are turned in the direction of limitation of movement. This leads to compensatory head posture comprising of face turn, head tilt and altered chin position mainly to alleviate diplopia. Usually symptom free in children with congenital palsies.

**Muscle Sequelae**

Determined by Hering's and Sherrington's laws. They are (1) Underaction of the affected muscle (2) Overaction of the contralateral synergist (Hering's law) (3) Contracture of ipsilateral antagonist (Sherrington's law) 4. Secondary inhibition of the contralateral antagonist (Hering's law).

These developments depend upon the duration of muscle palsy, degree of limitation of ocular movement and the eye which is fixing. Because of these muscle sequelae, the secondary deviation (the angle measured with the affected eye fixing) exceeds the primary deviation (the angle measured with the unaffected eye fixing).

# CLINICAL EVALUATION OF A CASE WITH OCULAR MOTOR NERVE PALSIES

After careful history including the medical and neurological conditions, the patient should be examined in general to rule out vascular diseases like diabetes, hypertension and arteriosclerosis. If there is history of cerebrovascular accident, a total neurological examination is needed to find out the associated neurological signs.

## Diplopia

If there is horizontal diplopia – Horizontal muscle palsy. As isolated medial rectus palsy is rare, it is most likely due to lateral rectus palsy. Vertical diplopia especially with a tilt, most likely to be superior oblique palsy.

## Cover Test

One should look for the type of deviation comparing the amount of movement for near and distance.

## Ocular Movements

Both underaction and overaction are to be noted by doing alternate cover test in different positions of gaze. Maximum deviation is noted in the direction of the action of affected muscle. The examiner should remember that both the eyes may be affected, particularly after severe head injury. Other signs like nystagmus especially in certain positions, or retraction of globe on horizontal or vertical gaze can occur in case of direct injury to muscles or their connection.

## Diplopia Chart

1. Maximum separation of images in the direction of action of the affected muscle.
2. Abductors – lateral rectus, superior and inferior obliques produce uncrossed diplopia while others produce crossed diplopia.
3. Tilt though produced in vertical palsies is more common with superior oblique palsies.

## Hess Chart

Not only diagnosis but also for follow up of a case of muscle palsy. Points to be remembered are

1. The smaller field always belongs to the paretic eye-inward displacement of dots indicates underaction of the paretic muscles and outward displacement indicate overaction of antagonist. Equal sized fields indicate muscle sequelae have developed denoting either congenital or longstanding palsy.
2. The outer field should be examined for small underaction and overaction which may not be apparent on inner fields. If the outer field is very close to the inner field, a mechanical restriction of muscle movement rather than a neurogenic one is suspected.

## Forced duction test and its applications

It is useful to differentiate a neurogenic palsy from restrictive palsy which may be due to contraction or fibrosis of a muscle, tightness of a muscle following excessive resection, scarring of conjunctiva, trapping of muscle fibres as in blow out fractures, symblepharon etc.

After application of local anaesthetic, the eye is grasped at the limbus with a forceps and is moved in the direction opposite to that in which mechanical restriction is suspected, taking care not to press the globe. When the test is done patient is asked to look at this hand which is held the direction in which the eye is moved. This is to prevent the influence of the patient's innervation which may otherwise counteract the passive movement of the globe simulating a mechanical restriction.

## Bielschowsky Head tilt test

To find out the vertical muscle involved, especially in cases of superior oblique palsies where on tilting, the eye becomes hypertropic on the affected side.

## THIRD NERVE PALSY

### Clinical Signs

In complete palsy, the following signs will be present

a. Ptosis of the affected side.

b. Eye divergent, may be slightly depressed.

c. Ocular movements – Limitation of adduction, elevation and depression especially on abduction will be present.

d. If ptosis is complete, there is no diplopia and hence there may not be any abnormal head posture.

e. Pupil may be dilated or normal. It is very important to note pupillary reaction since it may give a clue about both etiological and anatomical location of lesion. This is very important since pupillary involvement is one of the sign of aneurysm which is very dangerous. In longstanding palsies, signs of aberrant regeneration have to be noted.

## Superior division palsy

There will be underaction of superior rectus along with partial ptosis and this is mostly congenital. The affected eye is hypotropic with absence of elevation. The chin is elevated.

The muscle sequelae will be overreaction of contralateral inferior oblique, contraction of ipsilateral inferior rectus and secondary inhibition palsy of other eye superior oblique.

**Differential diagnosis of the condition are:**

1. Double elevator palsy: Usually congenital and nuclear.
2. Blowout fracture of orbit: With typical findings and history.
3. Thyroid myopathy: Especially of inferior rectus muscle.
4. Paralysis of vertical gaze (Parinaud's syndrome).

## Inferior division palsy

Very rare. As it contains the pupillary fibres, pupillary dilatation along with underaction of medical rectus, inferior rectus and inferior oblique muscles. As a result, the eye will be exotropic, intorted and hypertropic; face will be turned to the affected side and head tilted to the side of hypotropic eye.

Isolated single muscle involvements are very rare and mostly myogenic. But if there is medial rectus involvement an internuclear ophthalmoplegia is to be ruled out.

## TROCHLEAR NERVE PALSY

It supplies the superior obliques muscle. Commonest cause of palsy is trauma followed by ischemia.

It can be congenital or acquired. Being the longest and slenderest nerve emerging from dorsal aspect of midbrain it is more vulnerable to head trauma can be bilateral in severe head injury. Rarely involved in ischemic injury due to diabetes. Commonest symptom will be diplopia on looking down especially while climbing down the stair case.

### Clinical Signs

### Compensatory head posture

The chin is depressed and head tilted to the unaffected side. This position moves the eyes away from field of action of the superior oblique to overcome diplopia.

### Ocular Signs

The affected eye will be hypertropic & slightly convergent. Hypertropia will be more for near.

### Muscle Sequelae

There will be underaction of superior oblique, that is depression in adduction with overaction of inferior rectus of the opposite eye and the inferior oblique on the

same side. Diagnosis is by Bielschowsky head tilt test in which the hypertropia is greatest on looking to the affected side.

### Diplopia Chart

Shows uncrossed diplopia with maximum separation of images on **levodepression** in case of right superior oblique palsy.

### Hess Chart

According to muscle sequelae. Mostly found in outer segment if minimal.

### The binocular field

Will be displaced upwards to the affected side.

## SIXTH NERVE OR ABDUCENS NERVE PALSY

The sixth cranial nerve supplies the lateral recuts muscle and its palsy results in lateral rectus weakness. Though it may be different causes the most common cause is only idiopathic neuritis.

### Clinical Signs

Esotropia which is greater for distance than for near.

### Compensatory head posture

Face turn to the affected side in most cases. Sometimes, only for distant fixation.

## Muscle Sequelae

Overaction of the contralateral medial rectus, contracture of ipsilateral medial recuts and secondary inhibitional palsy of contralateral lateral rectus.

## Diplopia Chart

Uncrossed diplopia with maximum separation of images towards the affected side.

## Hess Chart

Inward displacement of the field in lateral rectus area while outward displacement of field in medial rectus area in other eye. Later on according to other muscle sequelae.

## Field of BSV

Displaced towards the unaffected side.

## EVALUATION OF NERVE PALSY

It forms one of the false localizing signs along with bilateral papilledema in cases of increased ICT. As the horizontal deviation can be overcome better than vertical it is usually evident in distant gaze. It is the only thing that is allowed apart from papilledema in cases of pseudo tumor cerebri. Anatomic localization of lesion in an isolated VI nerve palsy is difficult sometimes and may require CT & MRI in some cases. Tumors are more common in children if VI nerve palsy is not following post viral or post vaccination in children. Apart from routine CBC, ESR, collagen vascular studies, tests to rule out diabetes or HT, and thyroid (VI is very rarely involved in thyroid ophthalmopathy) and may even sometime require a Tensilon test

to clinch the diagnosis. CT and MRI are indicated if there is associated ICT and if there is no improvement even after 3 months of onset.

## ETIOLOGY &PATHOGENESIS

The sites of the lesions of various etiology may be in the nucleus, fascicle, subarachnoid space, cavernous sinus or orbit.

## OCULOMOTOR OR THIRD NERVE PALSIES

The oculomotor palsies are either of congenital or acquired type.

### Congenital

It is a rare, often unilateral palsy without any neurologic or systemic abnormalities generally. Usually due to perinatal trauma or hypoplasia of the nucleus or muscles. Pupillary involvement is variable, aberrant regeneration is common and divisional palsies may occur. Though rare it accounts for almost 50% of III nerve palsies in children. Slowly progressive occulomotor palsy is an indication for repeat MRI in spite of previous negative studies, as neurilemmoma can remain cryptic.

### Lesions of Oculomotor Nucleus

Nuclear lesion of III nerve is rare. Any nuclear lesion is characterized by irregularity, bilaterality, loss of parallelism and presence of diplopia.

### Causes

Vascular, neoplastic and lesser number of demyelinating toxic and inflammatory lesions are the most common causes of nuclear III nerve dysfunction.

Pupillary involvement in a nuclear lesion is variable. Based on these features, Daroff has formulated certain rules.

**Obligatory nuclear lesions**

a) Ipsilateral III nerve without contralateral superior rectus palsies with bilateral partial ptosis.

b) Bilateral III nerve palsy (without or with internal ophthalmoplegia) associated with spared levator function.

**1. Conditions which may be nuclear**

a) Bilateral total III nerve palsy.

b) Bilateral ptosis.

c) Bilateral internal ophthalmoplegia.

d) Bilateral Medial rectus palsy.

e) Isolated single muscle involvement (except LPS & SR).

**2. Conditions that cannot represent nuclear lesions**

a) Unilateral external ophthalmoplegia associated with normal contralateral superior recuts function.

b) Unilateral internal ophthalmoplegia.

c) Unilateral ptosis.

## Lesions of the Occulomotor Nerve Fasciculus

Any fascicular lesion may produce an ipsilateral palsy which may be total and often associated with pupillary involvement and cannot be differentiated from a palsy

caused by lesions outside brainstem but the topical diagnosis made by the coexistence of other neurologic signs which may present as any of the following syndrome.

## Causes

Intraparenchymal, vascular, neoplastic and demyelinating lesions

| Syndrome | Principle lesions | Signs | Site of Lesion |
|---|---|---|---|
| Nothnagel's | Tumor of pineal body | Ipsilateral III N palsy contralateral ataxia | Superior cerebellar peduncle |
| Benedict's | Bony tumors vascular lesions solitary tubercles Hemorrhages | Ipsilateral III N Palsy contralateral hemiathetosis | Red nucleus and tegmentum of midbrain. |
| Claude's | Thrombosis of the medial interpeduncular branch of the posterior cerebral artery | Ipsilateral III N Palsy contralateral ataxia and asynergia | Red nucleus and brachium conjunctivum. |
| Weber's | Occlusion of PCA gumma TB, tumor | Ipsilateral III N palsy contralateral paresis | Cerebral peduncle |

## Lesions of the oculomotor IV in the subarachnoid space

The lesions in the interpeduncular fossa may be located anywhere from its emergence from the brainstem to the point where the nerve penetrates the dura beside the posterior clinoid process to enter the cavernous sinus. It may present in 3 ways.

## 1. Ipsilateral pupillary dilation as a sole manifestation

Any compression of the oculomotor nerve from above and medial may produce only pupillary dilation. May progress to total III nerve palsy and hence to be followed closely. Most important lesions included – Aneurysms at junction of basilar and superior cerebellar artery and at head of basilar artery, tumors & Basal meningitis.

## 2. Oculomotor nerve palsy with pupillary involvement

Commonest form of presentation of III nerve paralysis. It may present as isolated palsy without any neurological signs.

The basal type of III nerve palsy may be

a. Isolated III nerve palsy without associated symptoms.

b. Associated with syndrome involving other nerves.

c. Syndrome of interpeduncular space – Bilateral III nerve palsies with bilateral hemiplegia.

## Causes

Vascular – Intracranial aneurysm arising at the junction of ICA and PCA is the commonest or aneurysm of basilar artery by direct compression or during rupture. Trauma – Either severe head injury or during surgery, tumour and other compressive

lesions. Infections – Basal meningitis usually in association with other cranial nerves including V, VI & VII nerves. Diabetes Mellitus – very rarely cause pupillary involvement and hence in every case of pupillary involvement an MRI or arteriography is indicated to the treatment of potentially dangerous cause of aneurysm.

## Kernohan Notch Syndrome

When there is a supratentorial pressure from a tumor or subdural hematoma, compressing III nerve as it crosses the tentorial edge, the pupillary fibres which are in the peripheral superior medial part of the nerve are affected first without significant involvement of other parts of III nerve. This is a neurological emergency and depending on this ipsilateral pupillary dilatation with or without contralateral hemiplegia ( due to involvement of cerebral peduncle) the site of burhole to be put is decided in neurological emergencies. Also known as acute or progressive clivus ridge syndrome.

## Isolated III Nerve Palsy with Pupil Sparing

Ischemic form the frequent cause of this type of palsy which includes – diabetes mellitus, systemic HT< atherosclerosis, migraine, temporal giant cell arteritis and SLE. Often associated with severe pain, presumably there is a central ischemic infarct that spares the more peripherally placed parasympathetic pupillary fibres, 40% of diabetic III nerve may involve the pupil. However, comprehensive lesions can cause III nerves palsy without pupillary involvement due to following reasons.

1. They grow so slowly that the pressure is evenly distributed sparing the relatively pressure resistant pupillary fibres.

2. Compression from below sparing the dorsally located pupillary fibres.

Rule of thumb is that an isolated oculomotor palsy with pupil sparing is diabetes mellitus and that an isolated oculomotor palsy with pupil involved is aneurysm at the junction of ICA and PCA. However, there are excitations. In general if the patient is over 50 and has complete oculomotor nerve palsy and a totally normal pupil arteriography as a routine is not recommended. If the III nerve palsy is incomplete, with or without pupillary involvement and with aberrant regeneration arteriogram is to be done without fail.

## Lesions of oculomotor nerve within the cavernous sinus

Usually it will be a polyneuropathy than an isolated involvement. Further divided into anterior cavernous sinus syndrome when V1 of V alone is involved and posterior cavernous sinus syndrome when both first and second divisions of V nerve are involved.

## It is characterized by

Partial ophthalmoplegia involving both III, IV & V nerves along with first or first and II division of V nerve.

Oculosympathetic paresis with small, poorly reacting pupil, which is pathognomonic or the pupil is usually spared.

May be associated with proptosis, edema of lids and congestion of conjunctivae.

Commonly leading to aberrant regeneration of III nerve.

May be associated with involvement of Optic nerve in anterior lesions.

## Major etiologic lesions are

| | | |
|---|---|---|
| Infections | : | Specific granulomatous infections like tuberculosis, syphilis, etc – Basal Meningitis. |
| Inflammatory | : | Giant cell arteritis, rheumatoid arthritis, SLE, Herpes Zoster, cavernous sinus thrombosis. |
| Vascular | : | caroticocavernous fistula, Intracavernous aneurysm of internal carotid artery, arteriovenous fistulae either spontaneous or traumatic. |
| Neoplastic | : | Metastatic tumors – common<br>Intrinsic tumors – multiple myeloma, extension from nasopharyngeal carcinoma.<br>Extrinsic tumors – Pituitary adenoma, craniopharyngioma, supra and parasellar meningioma. |
| Miscellaneous | : | Following the irritation of pituitary for extensive PDR, persistent primitive trigeminal artery. |

## Lesions of oculomotor nerve in the superior orbital fissure

This is always associated with IV and VI nerve involvement with or without II nerve involvement along with I division of V nerve. If it is associated with decreased vision due to II nerve involvement along with light proptosis it is called apical syndrome described later in painful ophthalmoplegia.

## Pseudoorbital apex syndrome

Results when large inter cranial masses like giant aneurysm, compressing the cavernous sinus and optic nerve to impede drainage from the orbit. The oculomotor palsy is incomplete involving either divisions of the nerve.

## Tolosa-Hunt syndrome

Idiopathic granulomatous inflammation of SOF leading to painful ophthalmoplegia which will be described later.

## Lesions of III nerve in orbit

As the nerve has divided into 2 divisions, partial III nerve palsy is frequent and in case of superior division involvement, it has to be differentiated from a nuclear lesion.

## Causes

Orbital tumors, contiguous sinus disease such as mucocele, tumor or infection. Infection may not be obvious or orbital trauma and orbital fracture.

## Ophthalmoplegic Migraine

Differentiating features from aneurysms are (1) Transient nature of ophthalmoplegia. (2) Recurrent rapidly clearing palsy (3) Frequent personal or family H/o migraine.

## Recovery from III nerve Palsy

4 possible mechanisms are present.

1. Complete recovery: Usually within 6 months – Noted in ischemic condition. However, aneurysms take nearly 2 years and traumatic cases from 6 months to 1-2 years.

2. No recovery or change in Palsy: Usually in cases where the nerve is transacted by trauma or chronic compression or has been infiltrated by a tumor.

3. Partial recovery: Especially if there is fascicular lesion.

4. Partial recovery characterized by oculomotor nerve synkinesis: Usually becomes apparent 9 weeks after injury.

## Aberrant regeneration of III nerve:

Often after trauma and aneurysms; occasionally after tumour or syphilis; Never after ischemic causes especially diabetes.

## Types

Primary – without apparent III nerve palsy in slow growing lesions of cavernous sinus such as meningiomas and aneurysm or birth trauma.

Secondary – following apparent III nerve palsy.

## TROCHLEAR NERVE PARALYSIS

Like III nerve, it may be either congenital or acquired.

## Congenital trochlear nerve palsy

Not very uncommon. Unmasked when on eye undergoes surgical correction. In adults, it may presented at $5^{th}$ or $6^{th}$ decade, when it decompensates after minor injury without any cause.

## Two diagnostic sings are:

1. Large vertical fusion amplitude (10-15 diopters).
2. FAT scan (Family Album Tomography) look at old photographs to detect longstanding head tilt indicative of congenital etiology.

## Acquired paralysis

Trauma is the commonest cause of acquired palsies which may be following a direct orbital, frontal or oblique cranial blows or a contrecoup contusion at the tentorial notch may be unilateral or bilateral. Though 68% of cases are due to trauma, the trauma may not be very severe at first instance – Mostly following a rear end automobile collision. Usually, there will not be any loss of consciousness and there will be just a bump on head without redness or swelling of eye. Usually recovers in 3 to 6 months.

## Lesions of the trochlear nerve nucleus

Lesions of trochlear nerve nucleus cannot be localized with certainty unless there are other neurologic signs suggesting intrinsic mesencephalic damage. It can be obscured by associated gaze palsy. It is due to either intrinsic lesions or extrinsic compression or dorsal mesencephalon, which may be due to vascular causes leading to hemorrhage, infarction, brainstem AV malformation, medulloblastoma, ependymoma or metastatic tumour.

Extrinsic lesions include – pinealoma, aqueductal stenosis, hydrocephalus associated with ipsilateral or contralateral III nerve palsy.

## Lesions of trochlear nerve fasciculus

Difficult to diagnose and differentiate from nuclear lesions but usually without any associated neurological lesion. Diagnosis is by neuroradiological investigations like CT or MRI. May be associated with contralateral Horner's syndrome as the sympathetic pathways run through the dorsolateral tegmentum of mesencephalon adjacent to trochlear fasciculus.

### LESIONS OF THE TROCHLEAR NERVE IN SUBARACHNOID SPACE

The nerve is susceptible to trauma or compression as it emerges from the dorsal surface of brainstem by avulsion of emerging rootless or contusion injury due to hemorrhage within the nerve. These are usually bilateral.

As the trochlear nerve passes forward the cavernous sinus, it is protected from trauma and compression from tumor or aneurysm by the overlying rigid margin of the tentorium. It can however be affected by neurological misadventure and also as part of meningitic process due to syphilis or tuberculosis or even due to minor trauma as this nerve is the thinnest cranial nerve. It can also be involved in cases of tentorial meningiomas, pinealoma, sphenoidal sinusitis, encephalitis, migraine and even Schwannomas. Schwannomas trochlear nerve palsies due to tumor may be associated with intracranial tumor and may need careful check up of ocular and neurological examination. Rarely it is involved in aneurysm in posterior fossa and also chronic mastoiditis and pseudotumor cerebri associated with papilledema.

## Lesions of Trochlear nerve within the cavernous sinus and superior orbital fissure

Usually involved with other cranial nerve palsies. However, isolated IV nerve palsy occurs in this area due to ischemic condition like diabetes or due to local granulomatous angiitis from Herpes zoster involving either division of IV nerve or even the geniculate ganglion. Rarely due to compression from persistent primitive trigeminal artery. Etiologies similar to III nerve.

## Lesions of trochlear nerve within the orbit

Commonest cause is trauma. May also be due to inflammation and ischemia but it is impossible to ascertain whether the damage has occurred to the nerve, trochlea or the superior oblique muscle and/or tendon. May also follow neurological and dental anaesthesia – probably due to ischemia. Superior oblique muscle can be affected due to Paget's disease or hypertrophic arthritis. Tenonitis, neoplastic infiltration and inflammatory process can also decrease the motility.

## Lesions of trochlear nerve of uncertain and variable location

It may be due to progressive sclerosis or allergic granulomatous angitis called Churg-Strauss syndrome. This is due to vasculitis involving the vasa nervorum of trochlear nerve.

Superior oblique myokymia is a rare phenomenon leading to oscillopsia. All cases appear to be benign. First reported by Hoyt. Cause is unknown and may respond to Tegretol.

Brown's syndrome which involves restriction of the muscle and sheath as it slides through the trochlea. It is not innervational but mechanical and it is therefore an entirely different problem.

## Recovery from IV Paralysis

1. Complete recovery – Following ischemia or closed head injury or after relief of compression form tumor or aneurysms.

2. Incomplete recovery – Leaving the patient with mild persistent vertical and torsional diplopia.

3. No recovery – Primarily after mesencephalic injury or with transection of the trochlear nerve by trauma or compression.

## ABDUCENS NERVE PARALYSIS

The $6^{th}$ cranial nerve is affected in 30-50% of all ocular muscle palsies. Lesions of the nerve produce different syndromes from those seen with oculomotor and trochlear nerve palsies. It may be either congenital or acquired.

## Congenital

Can ocular rarely following birth trauma and disappear in 6 weeks may be associated with horizontal gaze palsy which occurs as an isolated sporadic or a familial condition may be associated with other neurologic and/or systemic signs or present as syndromes. Two important syndromes are,

i. Mobius syndrome – Associated with other congenital malformations.

ii. Duane's syndrome – The exact etiology not known; not classified under neurologic palsies.

## Acquired Paralysis

Depending on the nature and location of lesion, the VI nerve may be involved separately or in association with other cranial nerves or neurological signs.

## Lesions of abducens nerve nucleus

As the abducens nucleus is close to the pontine horizontal gaze centre, a lesion here produces conjugate gaze palsy to the ipsilateral side while conjugate horizontal gaze is totally abolished in bilateral cases. In most cases it is associated with ipsilateral peripheral facial palsy as the facial nerve facial loops around the abducens nerve nucleus. This is often asymmetric and is worse in abducting eye because the abducens motor neurons are more vulnerable than those of the internuclear neurons. Common causes include ischemia, infiltration, inflammation, trauma and compression or a combination of several factors.

## Lesions of abducens nerve fascicle

When associated with gaze palsy it is difficult to differentiate from nuclear lesion. The common causes include –ischemia, tumour compression or infiltration, infection, inflammation especially dysmyelination.

## Syndromes associated with fascicular lesions

| Name | Site | Damage to | Lesion Produced |
|---|---|---|---|
| Foville's syndrome (or) syndrome of anterior – inferior cerebellar artery | Pontine tegmentum | 1. Facial & abducens nerve fascicles.<br>2. Nucleus of the tractus solitarius.<br>3. Central tegmental tract.<br>4. Spinal tract of trigeminal nerve and it s nucleus.<br>5. And/or superior olivary nucleus. | 1. Ipsilateral abduction paralysis.<br>2. Ipsilateral LMN facial palsy.<br>3. Loss of taste from anterior 2/3$^{rd}$ of tongue.<br>4. Ipsilateral Horner's syndrome.<br>5. Ipsilateral analgesia of face.<br>6. Ipsilateral peripheral deafness. |
| Millard Gubler's syndrome | Ventral paramedian pons | 1. Abducens fascicle.<br>2. Corticospinal tract.<br>3. And/or ventral part of facial fascicle. | 1. VI and VII cranial nerve palsies.<br>2. Contralateral hemiplegia. |
| Raymond's syndrome | Lower pons | 1. Abducens fascicle<br>2. Corticospinal tract. | Abduction palsy & contralateral hemiplegia. |

## Lesions of abducens nerve in the subarachnoid region

Because of the long course of the nerve, it is frequently involved due to varied causes in the subarachnoid region.

## The lesions include

**Vascular :** The basilar artery lies in between the two abducens nerves and the anterior inferior cerebellar artery claps around the nerve, along with posterior cerebellar artery which lies closer. The nerve may be compressed by the atherosclerosis, dolichoectasia or aneurysm of these arteries.

**Compressive lesions :** Space occupying lesions above the tentorium (transtentorial masses) posterior fossa masses, structural malformation like Arnold Chiari malformation.

**Trauma :** Either direct including neurological trauma or indirect from blunt trauma in closed head injury.

**Inflammation :** Meningitis – Bacterial, viral, syphilitic, tuberculous, neoplastic associated with AIDS; unilateral or bilateral VI nerve palsy along with other cranial nerve palsies are noted.

**Basal tumors :** Meningiomas and chordomas may produce abducens palsy without any other neurologic signs and symptom, frequently bilateral. Other tumors include subarachnoid trigeminal neuroma and exophytic spread of intrinsic posterior fossa tumors such as glioma, medulloblastomas or ependymoma. C.P. angle tumors like acoustic neuromas cause hearing loss and impairment of corneal reflex as first symptom along with VI nerve and VII nerve palsy and ataxia.

**Rise in ICP :** Compresses the nerve between the pons and basilar artery or stretches it along the petrous temporal bone leading to unilateral or bilateral VI nerve palsies. May also follow spinal anaesthesia or lumbar picture or myelography.

## Lesions of extradural portion of VI nerve at petrous apex

**Gradenigo's syndrome :** As the abducens nerve penetrates the dura overlying the clivus, it passes beneath the petroclinoid (Gruber's) ligament and close to mastoid air cells. In patients with severe mastoiditis, the inflammation extends to the tip of the petrous bone, producing local inflammation of meninges. There is VI and VII nerve paralysis along with hearing loss; and irritation of gasserian ganglion leading to severe ipsilateral pain in the face, around eye etc. associated with lacrimation, photophobia and diminished corneal sensation.

**Pseudo Gradenigo's syndrome :** Sometimes occur due to tumor infiltration at this region especially by nasopharyngeal tumor extension or compression due to aneurysms of intrapetrosal part of internal carotid artery.

Other causes include involvement of VI nerve following lateral sinus thrombosis or phlebitis extending into inferior petrosal sinus or due to dural arteriovenous malformation in the superior petrosal sinus or due to compression by the persistent trigeminal artery. Trauma leading to longitudinal fractures of petrous temporal bone may lead on to abducens palsy along with V, VI, VII nerve palsy with hemotympanum, Battle's sign and CSF otorrhea.

**Lesions of abducens nerve in the cavernous sinus and superior orbital fissure.** –same as that of III nerve involvement.

## Lesions of abducens nerve in the orbit

Due its short course in the orbit isolated involvement is rare. May be involved in orbital schwannoma or may be involved following dental anesthesia. It may be difficult to clinically differentiate the neural and muscular involvement of abduction.

## Lesions of abducens nerve of uncertain or variable location

Though it can occur in all ages, it is more common in children. Causes in children include a post vaccination viremia or in new born following nonspecific febrile illness or respiratory illness. Usually recovers within 10 weeks but may recur in the same eye.

# SYNDROME OF PAINFUL OPHTHALMOPLEGIA

This constitutes the combination of single or multiple oculomotor palsies with pain in and about the eye. They may present as various syndromes depending on the location of the lesion.

## Tolosa-hunt syndrome

It is an idiopathic nonspecific inflammation of cavernous sinus and pachymeningitis f the superior orbital fissure. The syndrome is self-limiting with dramatic response to corticosteroid therapy. It is characterized by sharp boring pain in and about the eye, ophthalmoplegia, sensory deficit in the first division of trigeminal nerve, dilated and fixed pupil, with optic nerve rarely involved. There may be even spontaneous remissions with complete or partial regression of deficits which may occur at intervals of months or years. Rarely bilateral.

## Superior orbital fissure syndrome of Rochon-Duvigneaud

Any lesion involving the orbit from the posterotemporal aspect leading to involvement of the three ocular motor nerves along with I division of trigeminal. May extend to cavernous sinus also.

Clinical features include slight exophthalmos, total ophthalmoplegia, severe pain, sensory loss in the distribution of I division of trigeminal and oculosympathetic fibres. Lesions: 1) Inflammation in the sphenoidal and ethmoidal air sinuses (2) tumors (3) Rheumatic periostitis (4) Aneurysm (5) trauma (6) periosteal hematoma due to trauma (7) bony changes (8) blood cysts.

## Orbital apex syndrome

Here along with ocular motor nerves, optic nerve, lacrimal and frontal nerves are involved. Ophthalmic artery with sympathetic plexus and orbital veins are also involved. Trauma leading to incomplete palsy and if due to hemorrhage it is reversible. May be involved in tumor invasion also where it may lead to compression of orbital vein – pseudoorbital apex syndrome.

## Cavernous sinus syndrome

The third, fourth, sixth along with sympathetic and ophthalmic trigeminal leading on to multiple ocular motor palsies pain, numbness along II division of trigeminal with "apparent pupillary sparing", VI nerve is most commonly affected as it forms content of cavernous sinus along with internal carotid artery.

## Divided into

Anterior cavernous sinus syndrome – III, IV, VI & V1 of V

Posterior cavernous sinus syndrome – III, IV, VI, V1 & V2 of V – described earlier.

## Diabetic ophthalmoplegia

20-45% of the mononeuropathies of III, IV & VI nerves are due to diabetes. In this, palsies of III & VI nerve are more frequent while the IV nerve is the least affected in diabetes. It can also lead to simultaneous involvement of two or more cranial nerves rarely. In these cases, CT & MRI are indicated to rule out compressive lesions. Pupillary sparing is an important feature of diabetic third nerve palsy as the lesion is mainly due to ischemic demyelination due to obstruction of the vasonervorum of the affected cranial nerve. Remyelination is thought to be responsible for recovery without aberrant regeneration.

## MANAGEMENT OF OCULAR NERVE PALSIES

Acquired cranial nerve palsy is symptomatic during the first few months, after onset. Diplopia in many patients is controlled by compensatory head posture and requires no treatment.

Some patients who are unable to control the diplopia the treatment options include the use of Fresnel prism's if the angle of deviation is small, uniocular occlusion and botulinum toxin injection into the uninvolved rectus muscle especially in lateral rectus muscle in case of oculomotor nerve palsy.

# AIM OF STUDY

- To find out the common etiology and incidence of the infra nuclear neurological lesions of III, IV and VI cranial nerves.
- To study the pattern of recovery in each type of nerve palsy in these cases.
- To emphasize the importance of non invasive techniques in these cases to clinch the localization of involvement and probable aetiology according to localization.
- To find out the mode of treatment to be followed in cases of both recent and established cases of neurological palsies involving III, IV and VI cranial nerves.

# MATERIALS AND METHODS

This study was conducted in department of ophthalmology, Coimbatore Medical College Hospital, Coimbatore. The study period was from august 2007 to august 2009. All patients attending to ophthalmology department and referred cases from other departments, who are having neurologic lesion of III, IV and VI cranial nerves mainly infra nuclear neuropathic ocular movement disorder were included in this study.

Patients with supra nuclear, nuclear, inter nuclear and myogenic types were excluded from the study by doing suitable examination and investigations.

Patients with any ocular cranial nerve palsies were indentified and their age, sex, duration of paralysis was noted. After this, a detailed history of the mode of onset, progression, and treatment if any received, or any previous surgeries was recorded. History or relevant associated diseases such as diabetes, hypertension, tuberculosis, syphilis, past meningitis, stroke, hypothyroidism, hyperthyroidism, myasthenia and migraine were noted. Then history specific to ocular complaints such as diplopia, blurring of vision, defective visual field, drooping of upper lid, vestibular complaints of vertigo, oscillopsia and tilt, were recorded. History of trauma, any history suggestive of raised intracranial tension, fever, ear discharge, and deafness was elucidated and recorded. Any other history that the patient has volunteered which was found relevant to the diagnosis was also noted. Patients were also questioned regarding family history of similar complaints and consanguinity. Personal history regarding smoking, alcohol intake and the diet history was also noted.

## GENERAL EXAMINATION

Each patient was subjected to a detailed general examination with particular reference to anaemia, generalized lymphadenopthy and nutritional status and blood pressure in adult patients.

## OCULAR EXAMINATION

Thorough ocular examination is done in all cases. Any abnormality in head posture, any head tilt, face turn, and chin position were noted. Any skull abnormalities also noted. The lids were examined for ptosis, retraction, lid lag, presence of Marcus Gunn phenomenon, or any other synkinetic movement. Amount of ptosis and presence or absence of levator action was also noted.

Regarding eye position, presents of deviation was first noted in all positions of gaze with either eye fixating. Ocular movement were tested in all positions in one eye (ductions) and also in both eyes (Versions and vergence). The primary and secondary deviations were tested by cover and un cover test.

A thorough examination of anterior segments using slit lamp was done. Pupils were tested for size, shape and reaction both direct and consensual, vergence and accommodation. Fundus examination was done meticulously to find out any associated fundus abnormalities.

The unaided visual acuity, retinoscopic value, corrected visual acuity, colour vision, examination of the fields- both central and peripheral as well as recording of intra ocular pressure were done as a routine.

Specific tests include Diplopia charting and Hess screen to find out both the defective muscle action also its sequelae. These tests were repeated during each review for the same reason.

## NEUROLOGICAL EXAMINATION

A full neurological examination was done on each patient. Examination of higher functions, other cranial nerves, motor and sensory systems and cerebellar functions were examined.

## INVESTIGATIONS

A complete haemogram, urine analysis, blood sugar estimation, Mantoux and Blood VDRL were done. X-ray of chest, orbit, paranasal sinuses and skull was done in indicated cases. CT scan and MRI were done in selected cases.

If necessary, neurologist's opinion, ENT opinion, diabetologist's opinion and any other specialist's opinion to come to definite clinical diagnosis was sought. After these appropriate treatment was started. Patient was asked to come for follow up once in two weeks.

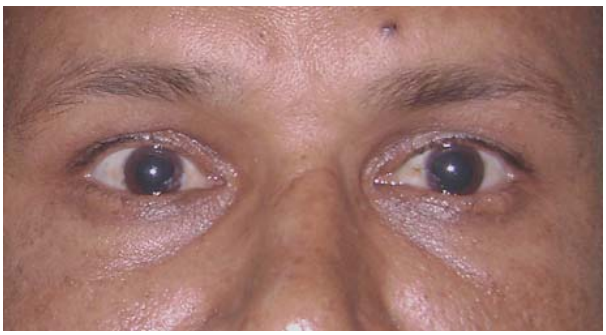
## TREATMENT

In all cases where a specific cause is found, such as diabetes, hypertension, tuberculosis or any other specific infection, the treatment was directed to the treatment of the cause. In cases of diabetic neuritis, diabetologist's for control and further usage of drugs was got.

For patients who had any specific neurological problems, opinion from neurologist was got and so also from ENT surgeon for suspected ENT causes. In case of non specific neuritis which proved to be the commonest cause, steroids in the form

of oral prednisolone in divided doses was given along with neuro vitamins and analgesics if there was associated pain. Steroids were tapered off after 6 weeks or in cases where there was partial recovery it was given for some more time in the same dosage and then tapered off.

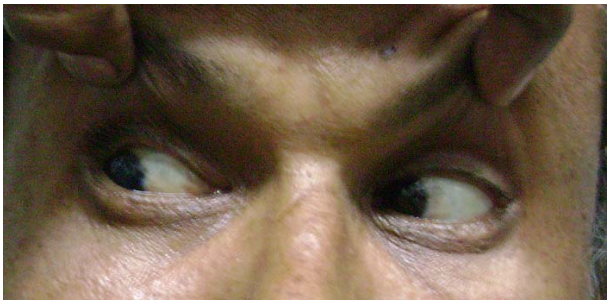
## Right Sixth Nerve Palsy



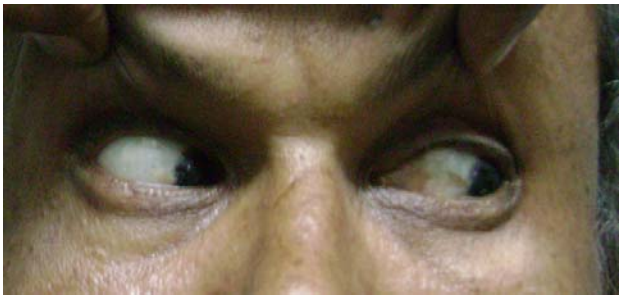
Primary Position

Limitation of right Abduction

## After Recovery



Normal Abduction



Normal Adduction

## Right Sixth Nerve Palsy

Slight Right Esotropia
in the Primary Position

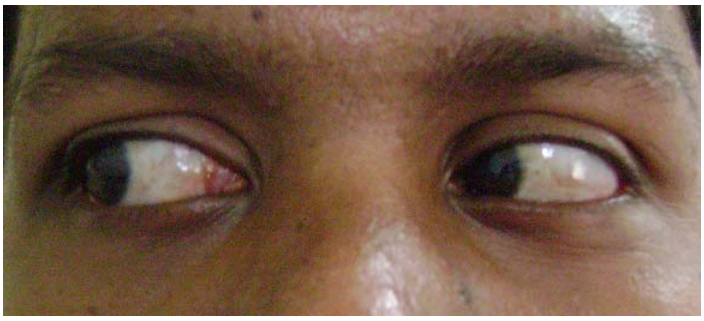
Normal Right adduction

Limitation of Right Abduction

Normal Elevation

## Recovery after Six Months

Primary Position



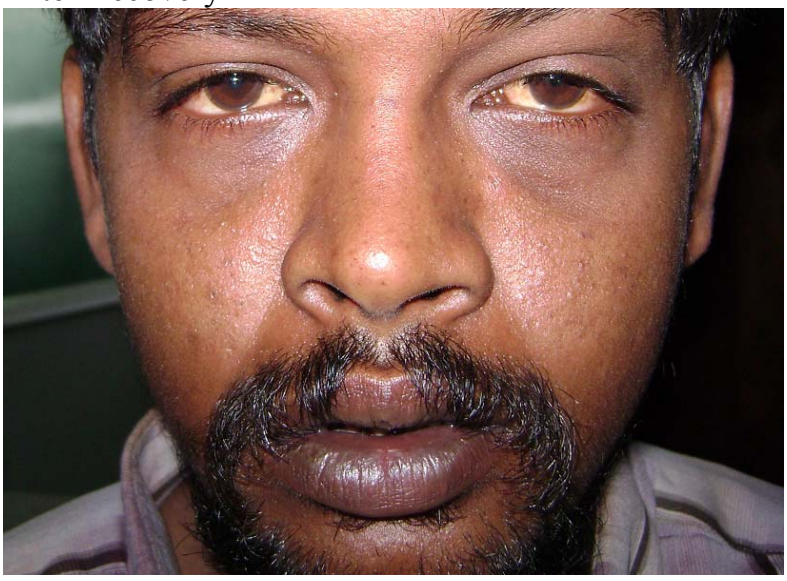
RE Normal Abduction

RE Normal Adduction

**Left Fourth Nerve Palsy**

Head Tilt to Right

After Recovery



Normal Head Posture

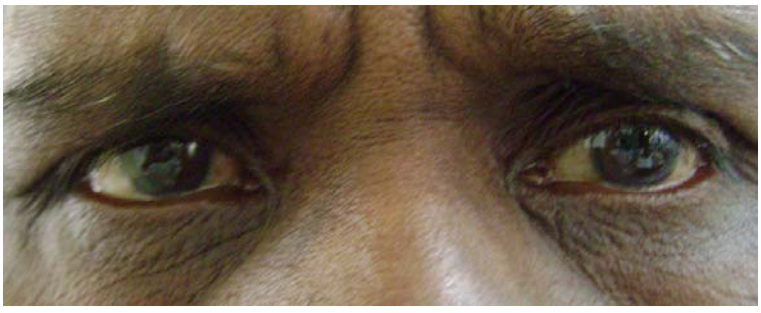
**Non-Recovered Right Sixth Nerve**

Primary Position
Right Esotropia

Absent Right
Abduction

Diabetic Partial Third Nerve Palsy

After Recovery



**RE III Nerve Palsy (Diabetic)**

RE Ptosis

Primary Position
RE Exotropic

RE Absent Adduction

RE Absent Elevation

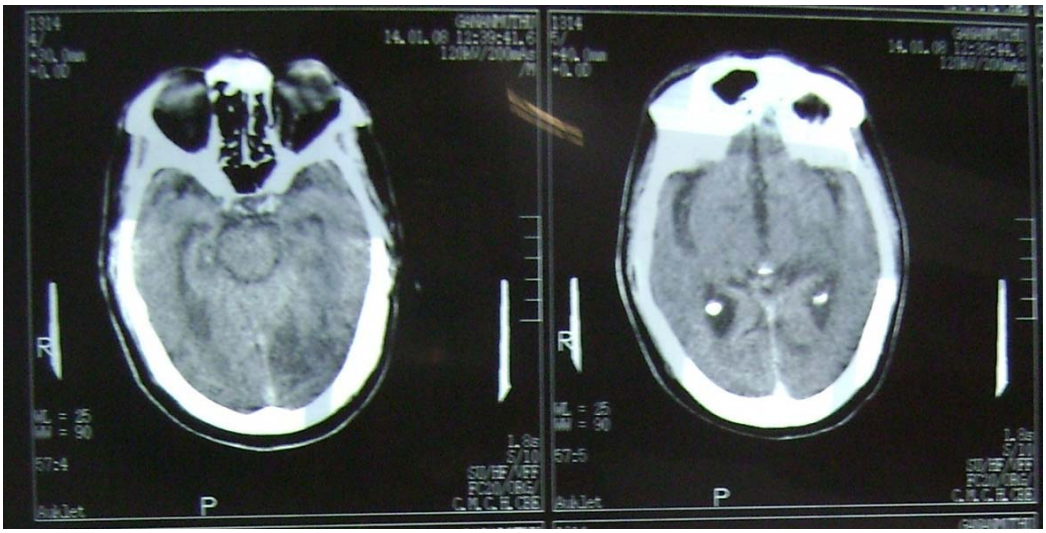
**Recovery After Three Months**

Primary Position

RE Normal Adduction

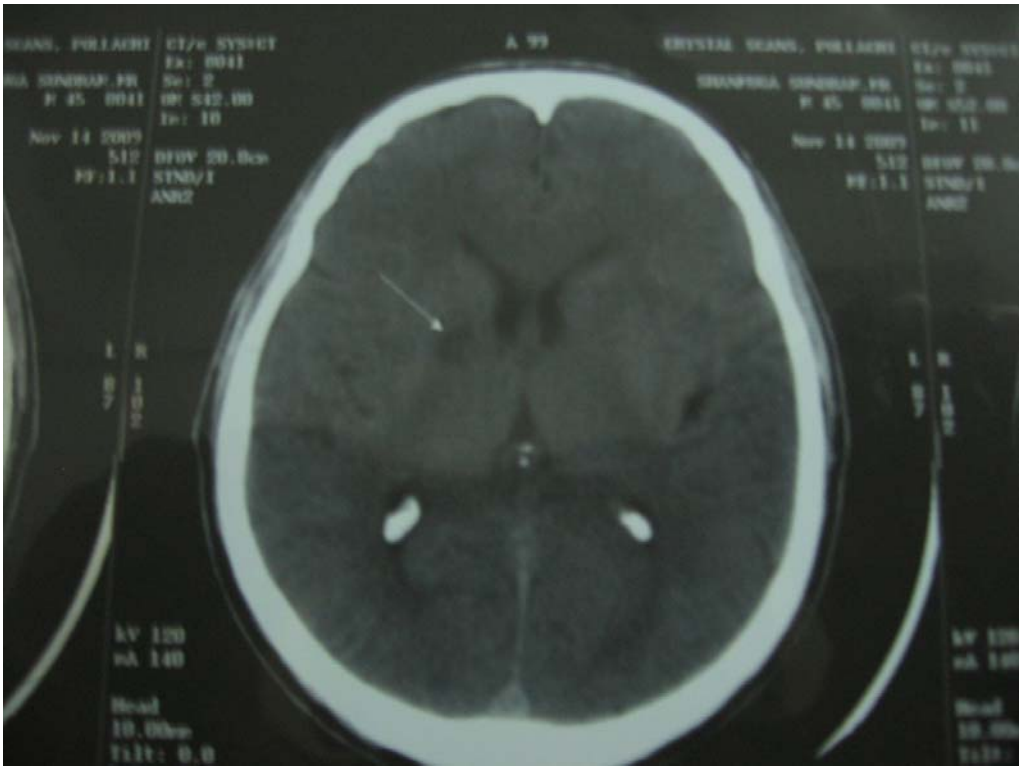
RE Normal Elevation

**CT-BRAIN**



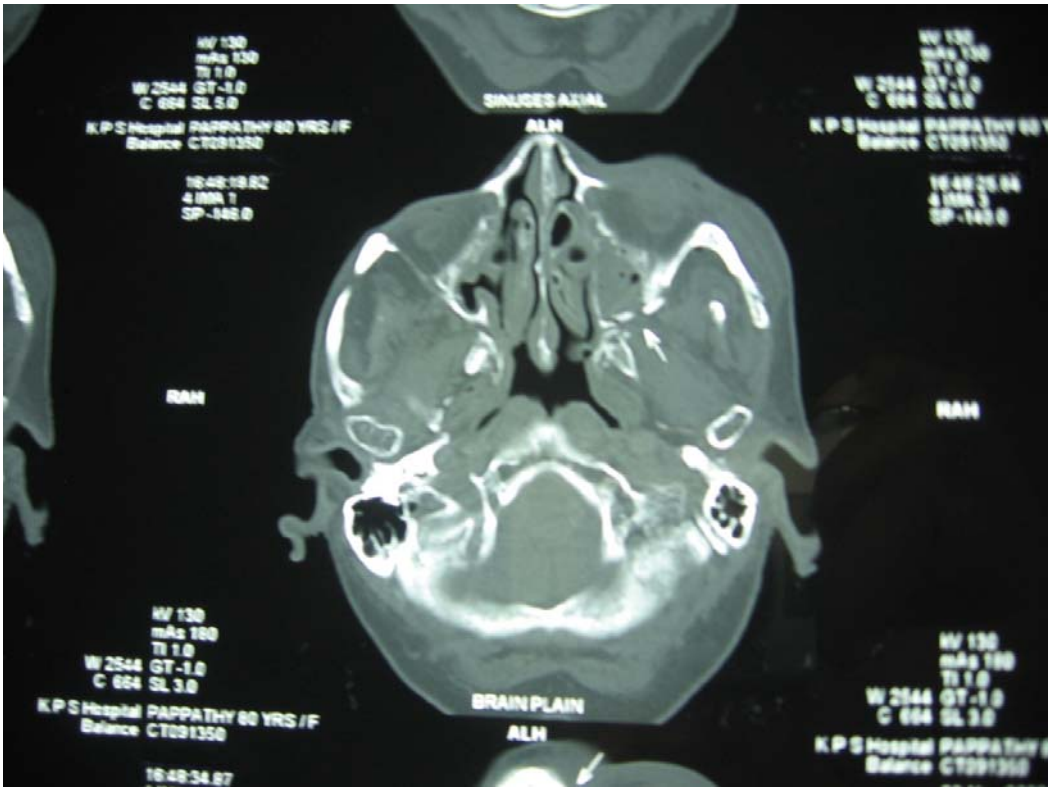
Non Hemorrhagic infarct in Left Half of Brain stem and Left Parieto Occipital Region

**CT-BRAIN**



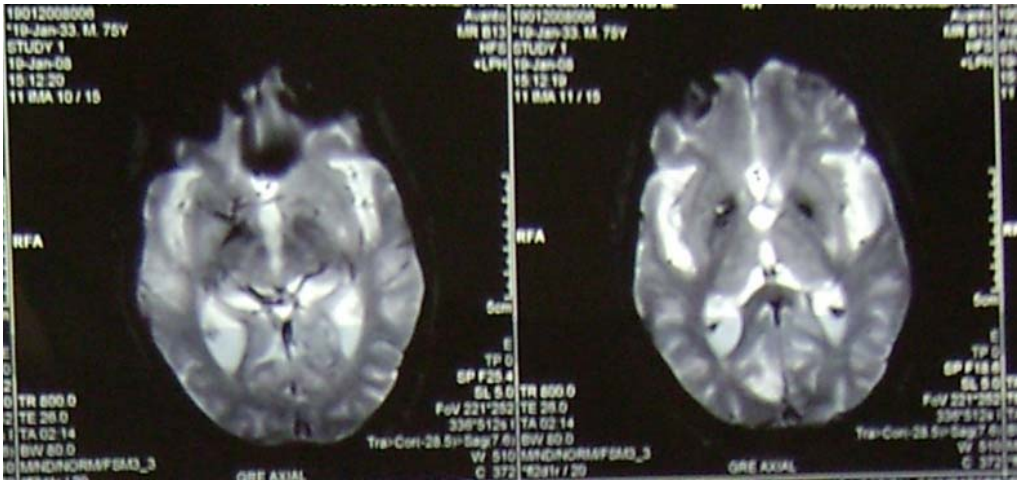
Right capsulo ganglionic infarct

**CT-BRAIN WITH ORBIT**



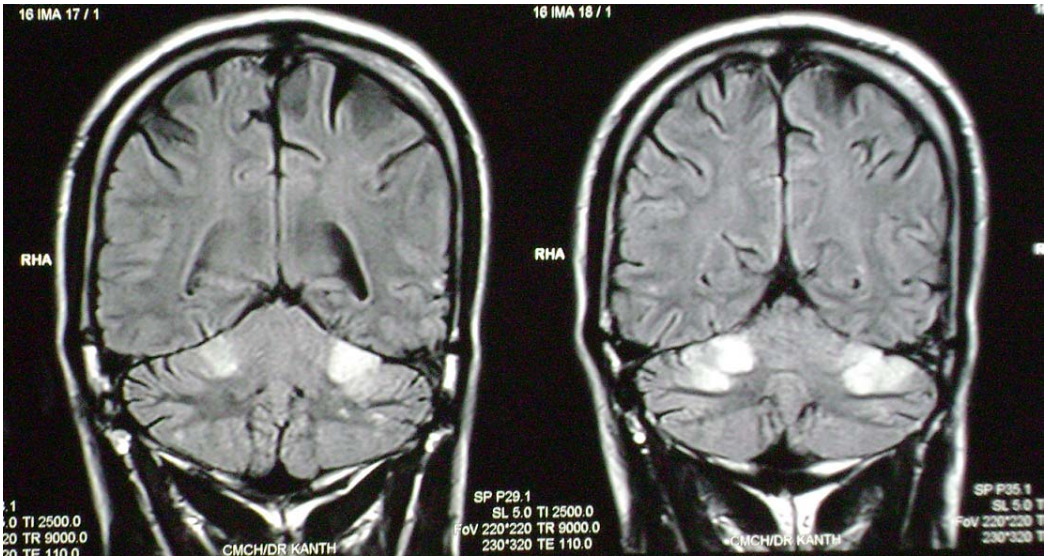

Fracture left frontal and temporal bone with pneumo encephalocele

**MRI-BRAIN**



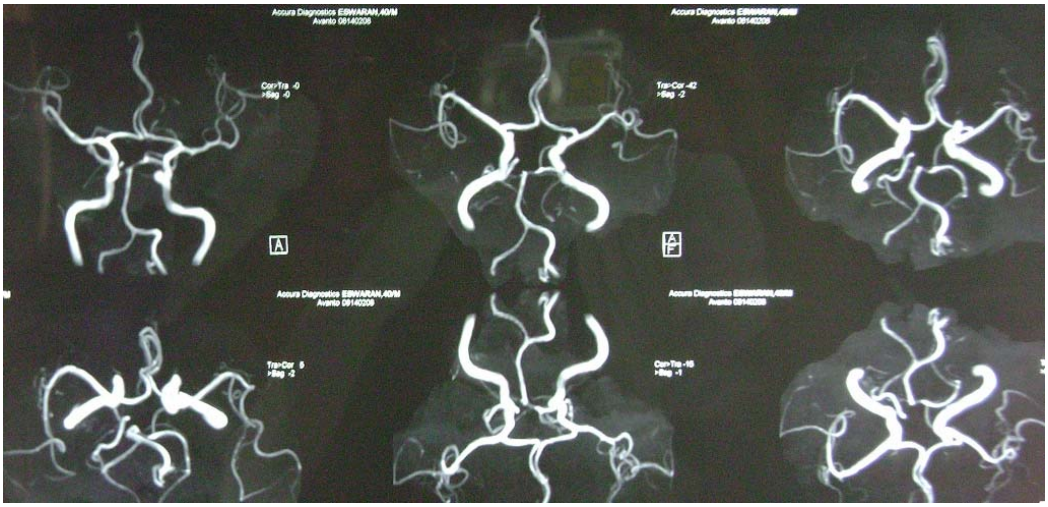

Sub-acute Infarct Involving Left Cerebellar Hemisphere, Brain Stem and Periventricular Ischemic Changes

**MRI-BRAIN**




Subacute infarcts in the posterior circulation involving the cerebellar hemispheres, brainstem and the medial thalami.

**MRI-BRAIIN**

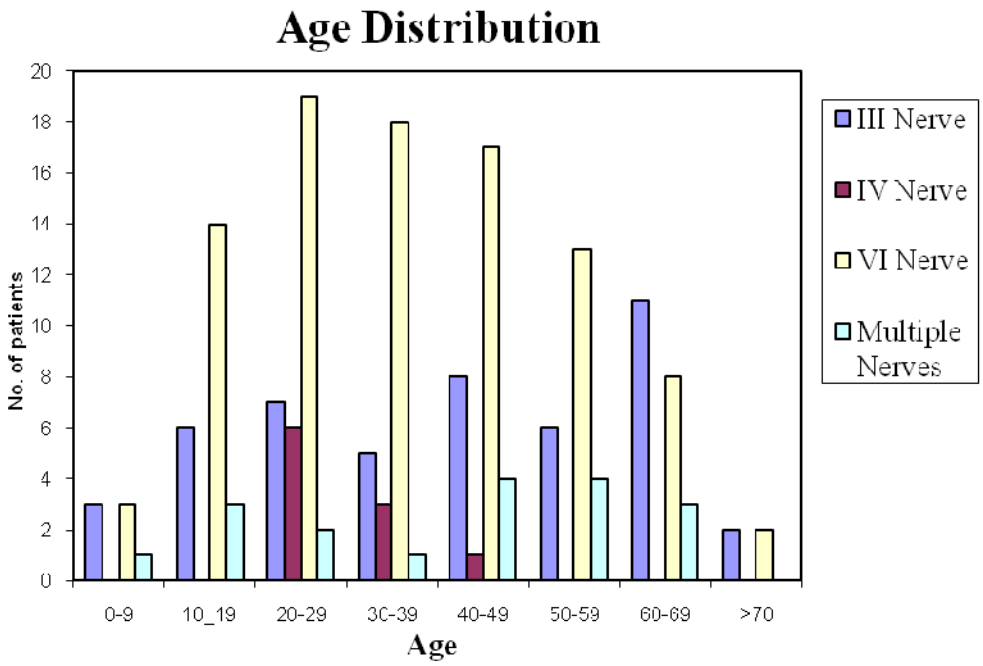


Normal MRA of Brain

# RESULTS

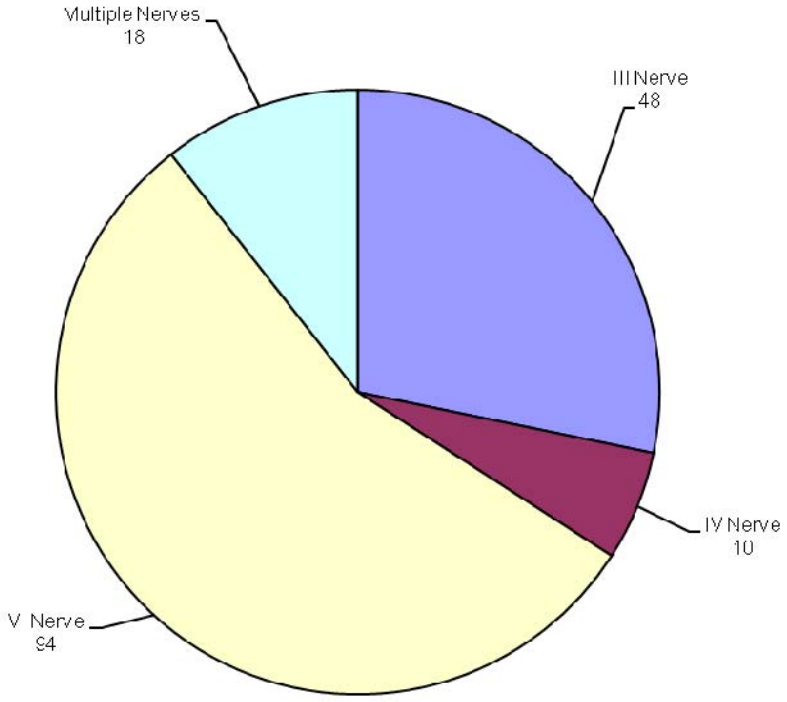
## AGE

| Age in years | III Nerve | IV Nerve | VI Nerve | Multiple Nerves | Total |
|---|---|---|---|---|---|
| 0-9 | 3 | - | 3 | 1 | 7 |
| 10-19 | 6 | - | 14 | 3 | 23 |
| 20-29 | 7 | 6 | 19 | 2 | 34 |
| 30-39 | 5 | 3 | 18 | 1 | 27 |
| 40-49 | 8 | 1 | 17 | 4 | 30 |
| 50-59 | 6 | - | 13 | 4 | 23 |
| 60-69 | 11 | - | 8 | 3 | 22 |
| >70 | 2 | - | 2 | - | 4 |
| Total | 48 | 10 | 94 | 18 | 170 |



Of the 170 cases of Ocular cranial nerve palsies, 82.35% occurred between 20-70 years, the range being 1 ½ - 70 years. In this study maximum number of III nerve palsies were found only between 60 – 69 years i.e. 11 cases (22.17%) followed by 40 – 49 years –8 cases (16.67%). All trochlear nerve palsies are between 20 – 44 years of age. There is wide distribution of cases of VI nerve palsies from 2$^{nd}$ to 7$^{th}$ decade, with 71.28% falling between 20 – 60 years of age. In case of multiple cranial nerve palsies 50% of the cases fell between 40 and 60 years of age. In case of III nerve palsies, 8 cases out of 10 cases due to diabetes and all 5 cases due to hypertension belonged to the age group between 45 to 70 explaining large number of cases in this age group who are prone for microangiopathic lesions due to these illnesses. In cases of IV nerve, trauma either closed head trauma or severe head injuries seemed to be the commonest cause involving adult males between 20 and 40 years.

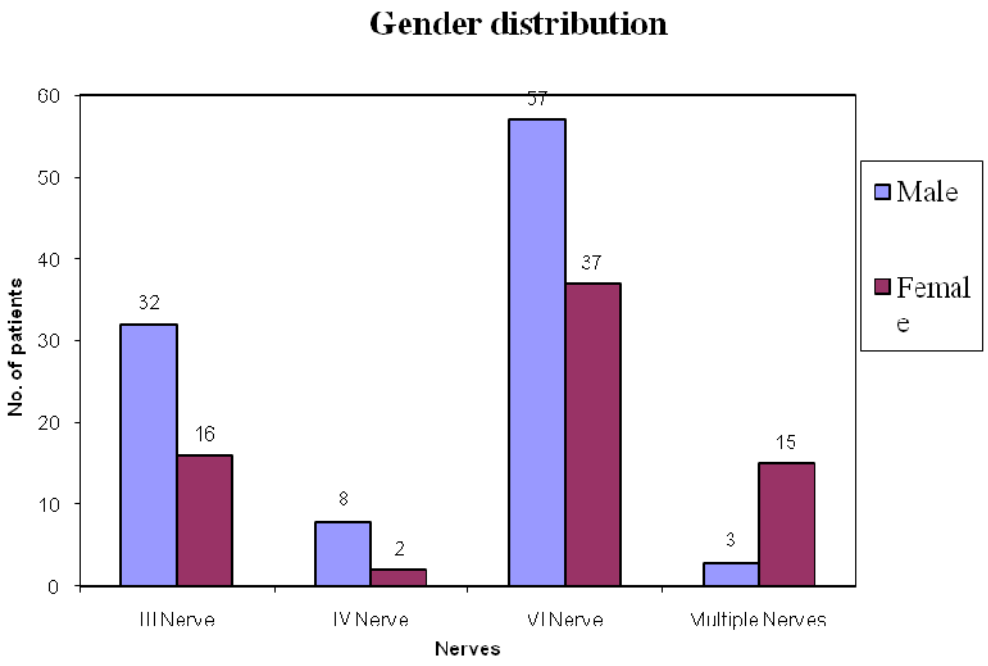
**Distribution of Ocular Cranial Nerve Palsies**



## GENDER

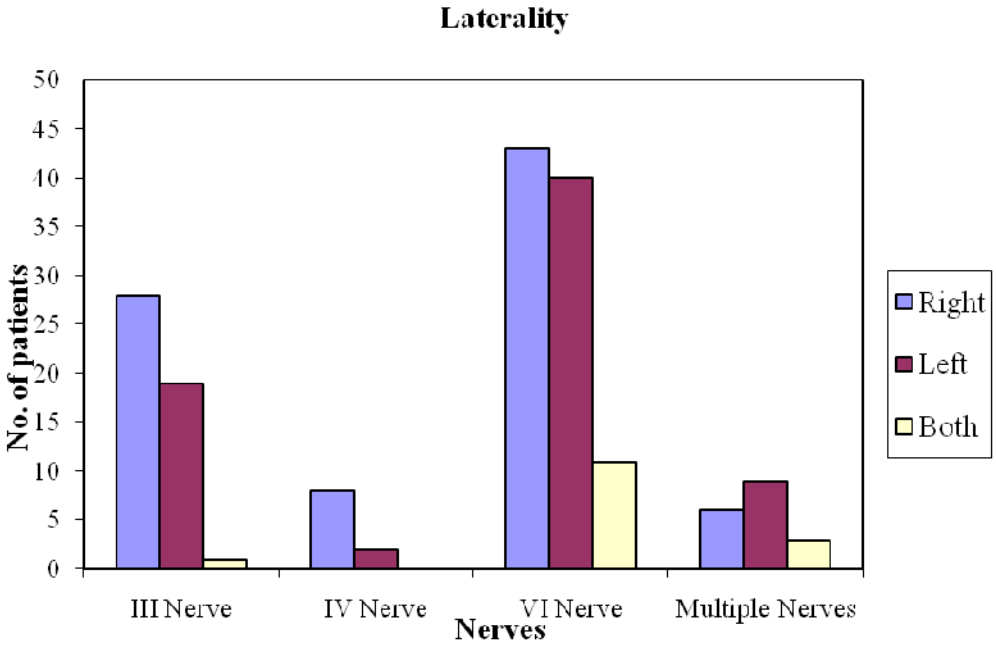
| Gender | III Nerve | IV Nerve | VI Nerve | Multiple Nerves | Total |
|---|---|---|---|---|---|
| Male | 32 | 8 | 57 | 3 | 100 (58.82%) |
| Female | 16 | 2 | 37 | 15 | 70 (41.18%) |

In general Males are more frequently affected i.e. 100 patients out of 170 (58.82%). In case of trochlear palsies 80% of the cases were found in males while in multiple cranial nerves affect more than 80% is in females.



LATERALITY

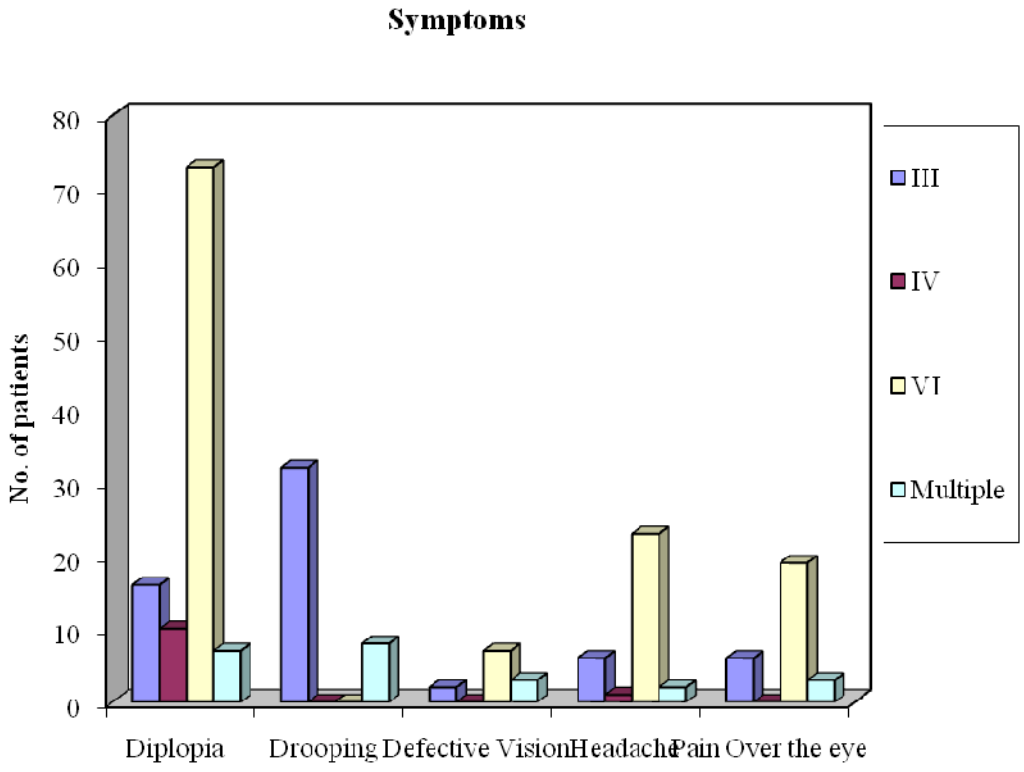
| Affected Eye | III Nerve | IV Nerve | VI Nerve | Multiple Nerves | Total |
|---|---|---|---|---|---|
| Right | 28 | 8 | 43 | 6 | 85 (50%) |
| Left | 19 | 2 | 40 | 9 | 70 (41.18%) |
| Both | 1 | - | 11 | 3 | 15 (8.82%) |




Right eye was the commonly affected eye i.e. in 50% of the cases except in cases of multiple cranial nerve palsies where half the number of cases occurred in LE while 16.66% occurred bilaterally and 33.33% affected in right eye. Of the 15 bilateral cases, one occurred in III nerve, 11 in VI nerve , and 3 with multiple cranial nerve involvement, , while there are no bilateral involvement with IV nerve palsies.

**SYMPTOMS**

| Nerve Affected | Diplopia | Drooping of Lid | Defective Vision | Headache | Pain Over the eye |
|---|---|---|---|---|---|
| III | 16 | 32 | 2 | 6 | 6 |
| IV | 10 | - | - | 1 | - |
| VI | 73 | - | 7 | 23 | 19 |
| Multiple | 7 | 8 | 3 | 2 | 3 |



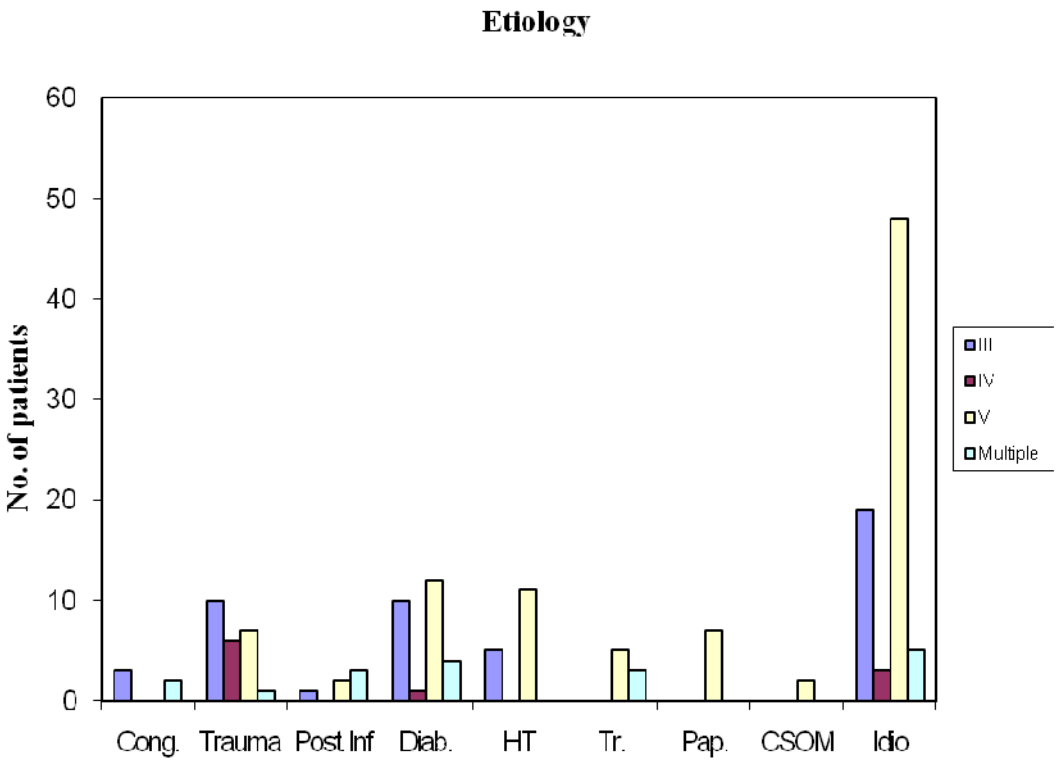
**General:** Commonest systemic association was diabetes Mellitus in 27 patients (15.88%) followed by fever in 14 patients (8.23%) Nausea and vomiting were found in few patients only. Most of the diabetes were aware of the disease and were under treatment. Only 4 had evidence of retinopathy.

**Ocular:** Commonest ocular symptom is diplopia especially almost in all cases of VI nerve and also in cases of IV nerve. In case of IV nerve reporting is mainly due to difficulty in climbing down stairs and reading. In patients with III nerve palsies the main complaint was drooping of the eyelid and diplopia was noted only during the recovery stage by many of them. In multiple cranial nerve palsies, depending on the involvement of nerves diplopia or drooping of lids formed the commonest presenting symptoms.

## AETIOLOGY

The various etiologies leading to affection of the ocular motor cranial nerves are given in the following tables.

| Nerve | Cong. | Trauma | Post. Inf | Diab. | HT | Tr. | Pap. | CSOM | Idiopathic |
|---|---|---|---|---|---|---|---|---|---|
| III | 3 | 10 (20.83%) | 1 | 10 (20.83%) | 5 | - | - | - | 19 (39.58%) |
| IV | - | 6 | - | 1 | - | - | - | - | 3 |
| VI | - | 7 | 2 | 12 | 11 | 5 | 7 | 2 | 48 |
| Multiple | 2 | 1 | 3 | 4 | - | 3 | - | - | 5 |
| **Total** | 5 | 24 (14.11%) | 6 | 27 (15.88%) | 16 | 8 | 7 | 2 | 73 (42.94%) |




## III Cranial Nerve Palsies

Of the total 48 cases, the largest number i.e. 19 belonged to non specific neuritis (39.58%). This diagnosis is arrived at after eliminating other possible causes by doing relevant investigations. Next is the microangiopathy due to diabetes – 10 cases (20.83%) and traumatic –10 cases (20.83%). 8 of the diabetic cases and the 5 hypertensive patients belonged to the age group between 45 and 70 years. In patients with Trauma only 2 people belonged to this age group while others were below 40 years where trauma is common.

## IV Cranial Nerve Palsies

Commonest cause is trauma i.e. 6 cases (60%). One diabetic (10%) and 3 non specific neuritis. (30%).8 cases in males – 6 of them due to trauma and 1 due non specific neuritis. All between 20 and 40 years.

## VI Cranial Nerve Palsies

Of the 94 cases more than 50% i.e. 48 cases (51.06%) belonged to non specific neuritis. Here too, all the relevant investigations and neurological examination were done to eliminate the other possible causes. 12 cases were due to diabetes followed by 11 cases due to hypertension, especially in older age group between 50 and 70 years. Two cases of children had CSOM associated with VI nerve palsy. Two cases one with III nerve and another with VI nerve involvement were thought to be due to demyelination after investigations and sent to neurology department for opinion.. 11 cases had bilateral VI nerve palsies. In these 4 cases had evidence of papilledema – 1 case following postpartum eclampsia improved well with treatment; 2 cases had hyperemic discs following fever with neck rigidity and were sent to paediatric neurology for further treatment; 1 case was due to uncontrolled hypertension in 31 years old male. In other cases, 1 was due to diabetes, 1 was due to non specific neuritis 1 was due to suspected sellar tumour and 1 suspected demyelination.

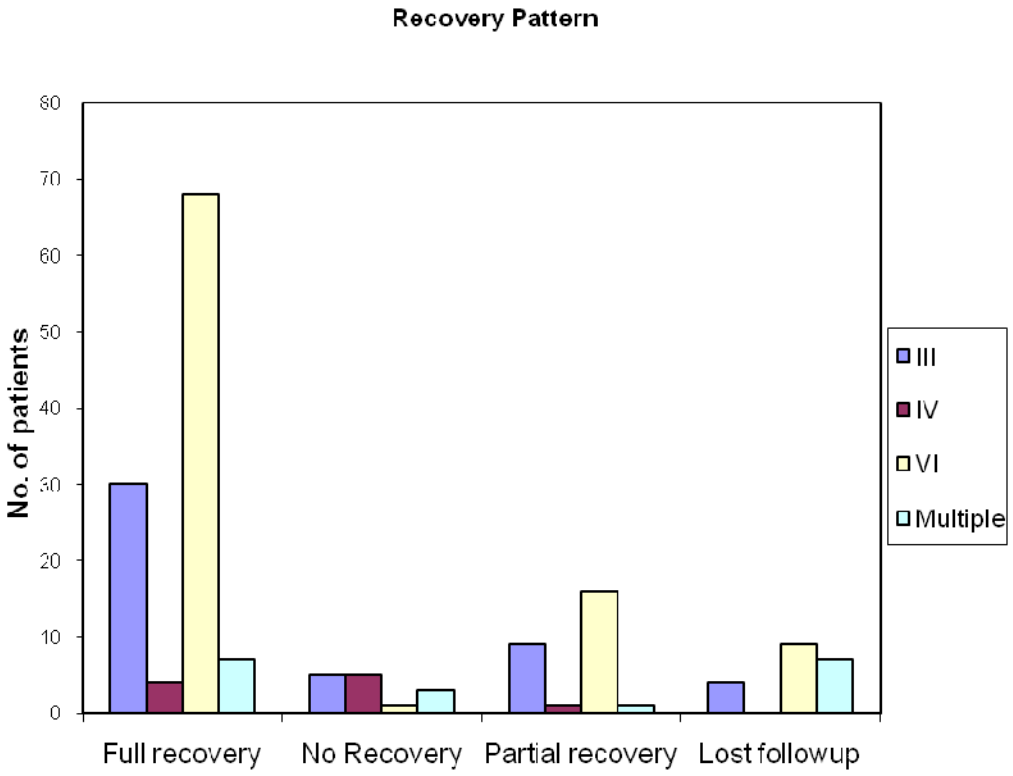
## Multiple Cranial Nerve Palsies

Of the 18 cases, 4 were due to diabetes, 1 following trauma, 3 following tumours, 3 following specific inflammation, 2 were congenital, 5 due to non specific inflammation. 3 cases of tumour were following astrocytoma, maxillary growth and juvenile nasopharyngeal angioma. 3 cases following specific inflammation were post Tuberculous meningitic, post typhoid, and post exanthematous fever

## RECOVERY PATTERN

All the ocular motor cranial nerves showed full recovery in more than 50% of cases but the recovery is poor if the lesion is due to trauma especially head injuries.

| Nerve affected | Full recovery | No Recovery | Partial recovery | Lost followup |
|---|---|---|---|---|
| III | 30 | 5 | 9 | 4 |
| IV | 4 | 5 | 1 | - |
| VI | 68 | 1 | 16 | 9 |
| Multiple | 7 | 3 | 1 | 7 |
| **Total** | 109 | 14 | 27 | 20 |



All the cases with non specific neuritis and also microangiopathy due to diabetes or hypertension showed mostly full recovery or at least partial recovery in 4 months. The average recovery time for IV nerve palsies due to non specific neuritis and diabetes ranged from 6 to 12 weeks. Average recovery time in VI nerve palsies ranged from 6 to 16 weeks. In III nerve palsies the recovery was noted within 4 weeks and the 1$^{st}$ muscle to recover was the LPS leading to diplopia if it was not present already. This was followed by medial rectus, superior rectus, inferior oblique and inferior rectus. Inferior rectus took longer time to recover. There was no discrepancy if the palsy was total or partial and age and sex had no correlation with the recovery pattern. Recovery was not good in posttraumatic cases. These were the cases along with those with organic lesions sent for higher investigations usually lost for followup.

# DISCUSSION

Ocular Cranial nerve palsies are always a diagnostic and therapeutic dilemma to the opthalmologists. The acquired cranial nerve palsies in this study are compared with similar studies done earlier to analyze the various etiologies and also general recovery pattern of these nerve afflictions.

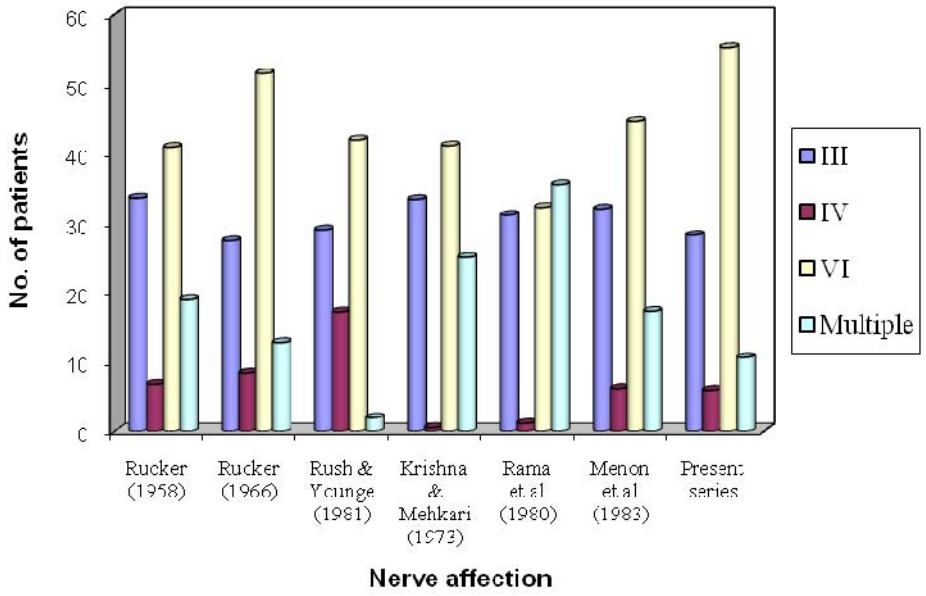
**Nerve Affection**

***Comparison cases of acquired extra ocular muscle paralysis Nerves Affected***

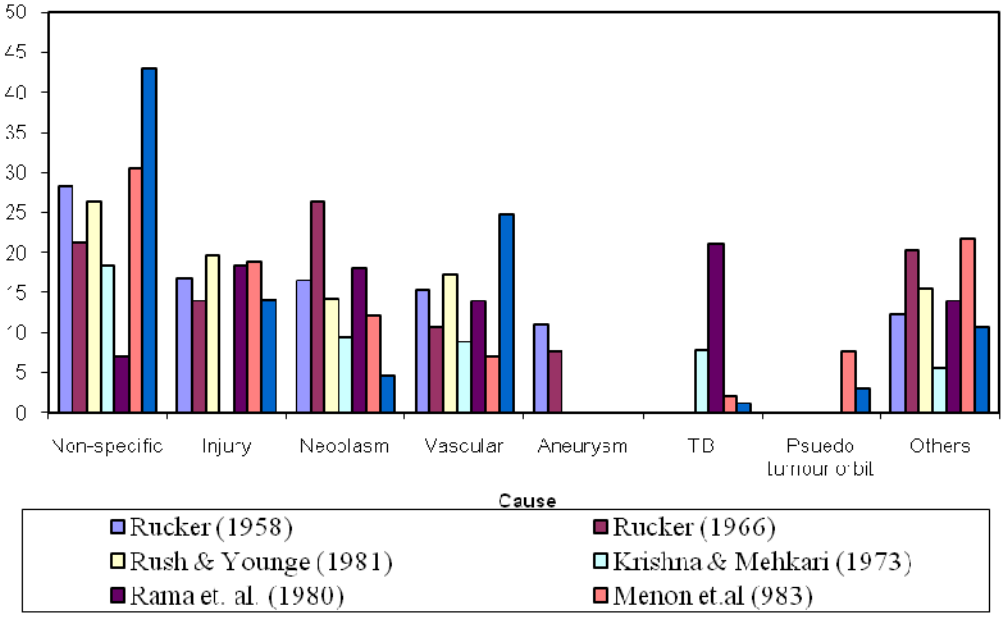
| Cranial Nerve | Rucker (1958) | Rucker (1966) | Krishna & Mehkari (1973) | Rama Et.al (1980) | Rush & Younge (1981) | Menon Et.al (1983) | Present series |
|---|---|---|---|---|---|---|---|
| III | 33.5 | 27.4 | 33.3 | 31.1 | 29.0 | 32.0 | 28.22 |
| IV | 6.7 | 8.4 | 0.5 | 1.1 | 17.2 | 6.1 | 5.88 |
| VI | 40.9 | 51.5 | 41.1 | 32.2 | 41.9 | 44.6 | 55.29 |
| Multiple | 18.9 | 12.7 | 25 | 35.5 | 1.9 | 17.3 | 10.59 |

In this study IV nerve was the commonly affected nerve (55.29%). This is in accordance with the studies quoted in the above table comparing both Western and Indian studies. As per the study conducted on 4298 cases of ocular motor paralysis by Richards, Jones and Younge from Mayo clinic in 1992, the largest group had involvement due to undetermined causes and the most frequently affected nerve was the sixth nerve. But according to the study by Berlit P in 1992 in a series of 412 patients the III nerve was the most commonly affected nerve. According to study by

**Comparison cases of acquired extra ocular muscle paralysis Nerves Affected**




**Causes of paralysis of cranial nerves comparison of previous reports and present series (%)**



Tiffin et.al also 57% were VI nerve but after that IV nerve in 21% while III nerve palsies is only 17%. VI nerve is more affected may be because of its long intracranial course with bends.

## Gender Distribution

In this study there is a slight pre ponderance of male patients (58.82%) slightly higher than compared with series by Rush (1981) and Richards Jones and Younge in (1992), where it 52% and 54% respectively. It may too probably due to the early approach of male patient for treatment and also an inherent hormonal protection in females. Laterality has got no significance in any of these studies.

## Presenting symptoms

Regarding presenting symptoms diplopia forms the commonest presenting symptom in 106 of 170 patients. (62.35%) same as that of the study by Kubutko-Zielinkska et . al in 1995 where diplopia formed the presenting symptoms in 90% of cases.

**Causes of paralysis of cranial nerves comparison of previous reports and present series (%)**

| Cause | Rucker (1958) | Rucker (1966) | Krishna &Mehkari (1973) | Rama Et. Al. (1980) | Rush & Younge (1981) | Menon et.al (1983) | Present series |
|---|---|---|---|---|---|---|---|
| Non-specific | 28.2 | 21.1 | 18.3 | 7 | 26.3 | 30.5 | 42.94 |
| Injury | 16.8 | 13.9 | - | 18.3 | 19.7 | 18.7 | 14.11 |
| Neoplasm | 16.5 | 26.3 | 9.5 | 18 | 14.3 | 12.2 | 4.70 |
| Vascular | 15.3 | 10.7 | 8.9 | 14 | 17.2 | 7.1 | 24.7 |
| Aneurysm | 10.9 | 7.7 | - | - | - | - | - |
| TB | - | - | 7.8 | 21 | - | 2.1 | 1.176 |
| Psuedo tumour orbit | - | - | - | - | - | 7.6 | 2.941 |
| Others | 12.3 | 20.3 | 5.6 | 14 | 15.4 | 21.75 | 10.72 |

Diabetes mellitus is the commonest general systemic disorder associated with ocular motor nerve palsies in correlation with the studies in the above table and also in the study by Watanabe et.al in 1990 who found out that the incidence of cranial nerve palsies in diabetic patients was significantly higher than in non-diabetic patients. 79/170 patients in this series were above the age of 40 where the incidence of diabetes is expected to be higher than in younger age group. In this study the incidence of diabetes was less than only to non specific causes. One case had pupillary involvement along with diabetes. As the success rate of CT increases above 60% according to the study conducted by Kwan ES et al, hypertension and diabetes

have to be ruled out before ordering CT and a time limit of about 2 to 4 weeks are allowed to note any improvement or worsening to decide about ordering higher investigative aids.

Trauma though commonest cause for trochlear nerve palsies, it can also affect the III and VI cranial nerves. According to the study by Tokuno T. Natkazawa K. et.al. there is high incidence of traumatic Sub Arachnoid Hemorrhage (71%) and skull fractures i.e. (57%). In this study only 14.70% are due to trauma but in these cases the recovery from the palsies is not good even after long followup in correlation with the above study.

## OCULOMOTOR PALSIES

### Congenital Palsies

In this series there were 3 congenital III nerve palsies. These were included in the study as the patients and parents were not sure of onset of problem and a diagnosis of congenital III nerve lesion was arrived at by excluding all other possible causes. Two cases reported in first decade and 1 in second decade. These cases were uniquely without any other neurological deficits unlike in the study by Tsaloumas and Willshaw HE in 1997 on congenital Oculomotor palsies where 5/12 patients had neurological abnormalities.

### Analysis of various etiologies

The results of this series is compared with the western and Indian series regarding etiologies in the above table. As in others non specific neuritis formed the commonest cause in the series also. This is followed by the vascular etiology due to diabetes and hypertension, which formed 24.7% of III nerve palsies in this series.

Here too comparing to that by Rush and Richards Younge, the largest group was non specific neuritis i.e. 39.58%. While vascular, trauma, aneurysm and neoplasms formed other common causes in their studies, there is no case of proved aneurysm in this series. Though pupillary dilatation was found in 6 cases, 4 of them are after trauma and one with diabetes and one with hypertension. In these cases apart from evidence of trauma the CT did not reveal any specific lesion. This is in comparison with the study conducted by Kwan ES et al., who stated that the overall diagnostic yield of CT for isolated III nerve palsies was low (30%) but improved to (60%) if diabetes and hypertension were excluded. It became highly sensitive i.e. 90% if associated with additional neurologic deficits. According to the study, the status of pupil in and of itself can not be the sole determinant as to whether angiography is indicated to exclude aneurysm. The study by JD Trobe in 1985 enumerates the conditions where arteography is indicated. According to the study by Bianchi Marzoli S, Brancato R. MRI has been confirmed to be the most important diagnostic tool, but relative non availability and high cost precluded its routine usage in this study.

## VI NERVE PALSIES

It formed the commonest occurring ocular motor palsy in most of the series and also in this series.

### Etiologies

Compared to the table above in this series also non specific neuritis form the major cause. According to the study by Miller et.al, vasculopathy followed by non specific neuritis formed the major cause.

## Laterality

In this study, RE affection is more than LE (58.23%) with 11 cases of bilateral involvement. According to the study conducted by Berlit. P et.al., it is frequent on left (52%) with 10% bilateral involvement. According to the study by Keane JR, this is almost equal to unilateral affection and these cases have to be thoroughly investigated. According to the study conducted by Galleta SL and Smith JL any chronic isolated VI nerve palsies have to be thoroughly investigated especially to rule out tumours. There were 5 cases of intracranial tumours mostly with papilledema and bilateral affection.

### Recovery

In this study 72% recovered within 3 months. In the study by King A.J et.al it is slightly higher i.e. 78.4%. In those who failed to recover nearly 40% had serious underlying pathology. In this series also patients who have not recovered had serious problems like tumour or traumatic causes.

# SUMMARY

1. Of the 170 cases of ocular cranial nerve palsies, 82.35% occurred between 20 – 70 years, the range being 1 ½ - 70 years. The maximum number of III nerve palsies were found only between 60 – 69 years. All trochlear nerve palsies are between 20 to 44 years of age. There is a wide distribution of cases of VI nerve palsies, from 2nd to 7th decade and multiple cranial nerve palsies 50% of cases fell between 40 – 60 years of age.

2. The distribution of cases is as follows:

   VI nerve palsies – 94

   III nerve palsies – 48

   IV nerve palsies – 10

   Multiple nerve palsies - 18

3. Number of patients affected.

   Male – 100

   Female - 70

4. Eye affected

   Right Eye - 85

   Left Eye – 70

   Both Eyes - 15

5. Most of the cases reported within 1 month of affection and generally followed upto 3 months at 2 weekly interval upto a maximum period of 4 months.

6. Commonest systemic association was Diabetes (15.29%) followed by fever.

7. Commonest ocular symptom was diplopia in VI and IV nerve palsies and drooping of eyelid in III nerve palsies.

8. In the 170 cases, aetiology could not be determined in 73 cases i.e. 42.94%.

9. Of the 48 cases of III nerve palsies, the largest number belonged to non specific neuritis. Next is microangiopathy due to diabetes and trauma – 10 cases each followed by five cases of hypertension.

10. Trauma formed commonest etiology in IV cranial nerve palsy occurring in 60% of cases followed by 3 cases of non specific neuritis one case due to diabetes.

11. In the 94 cases VI nerve palsies, 48 cases belonged to non specific neuritis. 12 cases due to diabetes 11 cases due to hypertension. 11 cases were involved bilaterally.

12. In case of multiple cranial nerve palsies, 3 were due to post inflammatory cause, 5 due to non specific neuritis, 4 due to diabetes, 1 due to trauma and 3 due to tumours.

13. There were 27 cases of Diabetes Mellitus (15.88%) and about 15% were diagnosed at the time of palsies. Only four cases had evidence of diabetic retinopathy. Pain or headache on the affected side was present in 82.6% of these patients.

14. Of the 15 cases with bilateral involvement, 11 cases were with VI nerve involvement. 3 cases with multiple cranial nerve involvement and one case of III nerve involvement.

15. Among the 25 cases following trauma, the injury ranged from trivial to severe head trauma and most of them are below 40 years of age.

16. There were 3 cases where there were recurrences. One was in a male with III nerve palsy who did not come to follow up regularly. One is in a female with IV nerve – non specific neuritis and other in male in right eye VI nerve with H/O diabetes under treatment.

17. 109 cases had full recovery and another 27 cases had partial recovery.

18. Post traumatic cases had fewer chances for full recovery. The dropouts in follow up especially after 1 or 2 months belonged to this group.

# CONCLUSION

From this study of 170 cases of cranial nerve palsies it is concluded that.

1. The VI cranial nerve is the most commonly affected nerve followed by III nerves and multiple cranial nerves and lastly the IV cranial nerve.

2. All these Ocular motor nerves can be affected by various pathological conditions.

3. Commonest presenting symptoms were diplopia, drooping of eye lid, pain and headache especially on the affected side.

4. Among the systemic conditions, diabetes followed by hypertension form the most commonly occurring association.

5. Closed head trauma even trivial is associated with trochlear nerve palsies while forcible head trauma as in cases of RTA leading III & VI cranial nerve palsies also.

6. There is no specific preponderance of infection in this series.

7. In 73 cases no specific cause could be made out

8. patients belonging to older age group were suffering from ocular motor cranial nerve palsies due to micro angiopathic lesions while trauma was the main cause in younger age group;

9. Non invasive procedures like CT & MRI were invaluable in diagnosis in selected cases.

10. Cases due to non specific neuritis or due to micro angiopathic lesions secondary to diabetes, hypertension etc recovered with in 4 months regardless of the nerve affected.

11. Longer follow-up, better facilities to do MRI may help in finding out specific aetiologies in these cases classified as nonspecific neuritis.

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# COIMBATORE MEDICAL COLLEGE HOSPITAL

## Department of Ophthalmology

### *Proforma*

### "A Clinical Study of Ocular Motor Nerve Palsies"

**1.** Name:_____

**2.** IP No: _____Date:_____

**3.** Age: _____

**4.** Sex: a) Male [ ], b) Female [ ],

**5.** Educational Qualification: No formal Education[ ], Schooling[ ], UG[ ], PG and above[ ]

**6.** Occupation: Studying [ ], Govt [ ], Private [ ], Own Business [ ], House wife [ ], Not working [ ],

**7.** Income:_____

**8.** Address: C/o,_____

Door No: _____

Street: _____

Place: _____

City: _____

PIN: _ _ _ _ _ _ _ _ _ / Phone: _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _

**9.** Referred from: _____

**10.** Presenting Complaints: _____

**11.** Presenting Illness:

| | |
|---|---|
| H/o Diminution of Vision | [ ] |
| H/o Double Vision | [ ] |
| H/o Deviation of eyes | [ ] |
| H/o Drooping of Lids | [ ] |
| H/o Photophobia | [ ] |
| H/o Watering | [ ] |
| H/o Fever | [ ] |
| H/o Head ache | [ ] |
| H/o Vomiting | [ ] |
| H/o Disturbance in Smell | [ ] |

H/o Deviation of Mouth [ ]
H/o Hearing disturbance [ ]
H/o Tinnitus [ ]
H/o Vertigo [ ]
H/o Difficulty in swallowing [ ]
H/o Disturbance in taste [ ]
H/o Loss of Weight [ ]
H/o Loss of Appetite [ ]
H/o Bowel and bladder disturbance [ ]
H/o Trauma [ ]
H/o Contact with Pet Animals [ ]
H/o Ear discharge [ ]

12. Past History:

| | Yes | No | Duration |
|---|---|---|---|
| Diabetes Mellitus | [ ] | [ ] | [ ] |
| Hypertension | [ ] | [ ] | [ ] |
| Tuberculosis | [ ] | [ ] | [ ] |
| Seizure disorder | [ ] | [ ] | [ ] |
| Medical History | [ ] | [ ] | |
| Surgical History | [ ] | [ ] | |

If Yes, Details: _____

13. Personal History:
    a. Diet: Veg: [ ] Non Veg: [ ] Mixed: [ ]
    b. H/o Consumption of Alcohol: Yes [ ] No: [ ] If Yes:
       Duration:_____
    c. H/o Smoking: Yes [ ] No: [ ] If Yes:
       Duration:_____
    d. H/o Chewing: Yes [ ] No: [ ] If Yes:
       Duration:_____
    e. H/o Drug intake: Yes [ ] No: [ ] If Yes:
       Duration:_____

14. General Examination :

a) Consciousness [ ]

b) Orientation [ ]

c) Higher functions [ ]

d) Built: Mild [ ], Moderate [ ], Well [ ]

e) Nourishment:Ill [ ], Moderate[ ], Well[ ]

f) Anaemia [ ]

g) Jaundice [ ]

h) Cyanosis [ ]

i) Clubbing [ ]

j) Generalized lymphadenopathy [ ]

15. Blood Pressure: _____/_____ mm of Hg.

16. Pulse Rate: _____/ Min.

17. Eye Examination:

a) Head Posture : Normal: [ ] Abnormal: [ ]

_____

b) Facial Symmetry : Normal: [ ] Abnormal: [ ]

_____

c) Eye: : Right Eye: Left Eye:

i. Vision: _____/_____

ii. With correction: _____/_____

iii. Eye Brows: _____/_____

iv. Lids: _____/_____

v. Conjuctiva: _____/_____

vi. Cornea: _____/_____

vii. Anterior Chamber:_____/_____

viii. Iris: _____/_____

ix. Pupils:

a. Size: _____/_____

b. Shape: _ _____/_____

c. Reaction to Light:

Direct: _____/_____

Consensual: _____/_____

d. Reaction to Accommodation: _____/_____

x. Lens: _____/_____

xi. Proptosis: _____/_____

xii. Nystagmus: _____/_____

d) Movement of the Eye ball:

Elevation : _____/_____

Depression : _____/_____

Abduction : _____/_____

Adduction : _____/_____

Intorsion : _____/_____

e) Intra Ocular Pressure: _____/_____mm of Hg

Schitz

f) Fundus:

Media : _____/_____

Disc : _____/_____

Vessels : _____/_____

Macula : _____/_____

Other findings : _____/_____

g) Fields: _____

h) Colour Vision:_____/_____

i) Diplopia Charting:

| | | |
|---|---|---|
| | | |
| | | |
| | | |

j) Hess Chart:

18. Other Systems:

1. Cardio Vascular System: S1 S2 Heard
2. Respiratory System: Normal Vesicular Breath Sounds Heard.
3. Abdomen: Yes No

Soft: [ ] [ ]

Organomegaly: [ ] [ ]

| | | | Normal | Abnormal |
|---|---|---|---|---|
| 4. | Central Nervous System: | | | |
| | i. | Other Cranial Nerves | [ ] | [ ] |
| | ii. | Motor System | [ ] | [ ] |
| | iii. | Sensory System | [ ] | [ ] |
| | iv. | Extra Pyramidal System | [ ] | [ ] |
| | v. | Cerebellar Functions | [ ] | [ ] |
| 5. | Examination of Ear, Nose, Throat: | | [ ] | [ ] |
| 6. | Examination of Thyroid: | | [ ] | [ ] |
| 7. | Examination of Breast: | | [ ] | [ ] |

19. Investigation:

BLOOD

Sugar :

Hb $_{A1C}$ :

T.C :

D.C :

E.S.R :

VDRL :

Mantoux :

URINE: _____

X-ray Skull: _____

X-ray Orbit: _____

C.T.Brain: _____

C.T.Orbit: _____

M.R.I: _____

Other Investigations:

_____

Referred to: _____

20. Provisional Diagnosis:

_____

21. Treatment:_____

Follow up:

# ABBREVIATIONS

| | | |
|---|---|---|
| RE | - | Right Eye |
| LE | - | Left Eye |
| Dip | - | Diplopia |
| Drop | - | Drooping |
| Def.Vn | - | Defective vision |
| Def.Movt | - | Defective movement |
| H/A | - | Headache |
| IMC | - | Immature Cataract |
| Mat Cat | - | Mature Cataract |
| A | - | Acting |
| NA | - | Not Acting |
| POA | - | Primary Optic Atrophy |
| HT | - | Hypertension |
| Gr. | - | Grade |
| Pap | - | Papilloedema |
| PT | - | Pulmonory Tuberculosis |
| ONH | - | Optic Nerve Head |
| # | - | Fracture |
| FR/PR/NR | - | Full/Partial/No recovery |
| LF | - | Lost follow up |
| SOF | - | Superior Orbital fissure |
| ATT | - | Anti tuberculous Treatment |
| RTA | - | Road Traffic Accident |
| LOC | - | Loss of Consciousness |
| PRE. SYM | - | Presenting Symptoms |
| ASS. SYS.DIS | - | Associated Systemic Diseases |
| F | - | Fundus |
| CT | - | Computerised Tomography |
| DEMYELI | - | Demylination |
| BDR | - | Background Diabetic Retinopathy |
| BS | - | Blood Sugar |

# MASTER CHART

| Sl No. | Age | Gen | Lat | Sym & Duration | Ass.Sys.Dis | V/A | A.S | Pupil | Fundus | Nerves | CNS | ESR-mm/hr | B.S | MX | X-ray | CT/MRI | Followup |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 22 | F | RE | Dip-1MT | - | 6/12 | N | A | N | VI | NAD | 12 | 90 | -Ve | - | - | 4 months-FR |
| 2 | 60 | M | RE | Drop-3D Pain-3D | DM | 6/36 | IMC-BE | A | NV | III | NAD | 11 | 240 | -Ve | NAD | - | 3 months-FR |
| 3 | 13 | M | RE | Dip-1MT | RTA | 6/6 | N | A | N | VI | NAD | 5 | - | - | # base of skull | - | 4 months-FR |
| 4 | 15 | M | LE | Drop-3D | H/O Injury | 6/6 | N | NA | N | III | NAD | 12 | - | -Ve | NAD | NAD | 3 months-FR |
| 5 | 59 | M | RE | Dip-1D Def VN-1D | HT | 6/9 | N | A | Gr.I HTR | III | NAD | 10 | 100 | -Ve | NAD | NAD | 4 months-FR |
| 6 | 53 | M | LE | Dip10 D | - | 6/6 | N | A | N | VI | NAD | 8 | 110 | -Ve | NAD | NAD | 3 months-FR |
| 7 | 35 | F | BE | Dip H/A15D | PP eclampsia | 6/6 | N | A | BL. PAP | VI BE | NAD | 5 | 81 | -Ve | NAD | Enlarge-Ventricle | 3 months-FR |
| 8 | 11 | M | RE | DipH/A-10D | Fever/Vomiting | 6/6 | N | A | N | VI | NAD | 14 | 60 | -Ve | NAD | NS | 4 months-FR |
| 9 | 50 | F | LE | Defvn Dip 15D | Nil | 6/60 | IMC-BE | A | N | VI | NAD | 10 | 74 | -Ve | NAD | NS | 4 months-FR |
| 10 | 55 | F | LE | Defvn Dip I Month | Nil | HM | MC | A | NB | VI | NAD | 16 | 85 | -Ve | NAD | NS | 4 months-FR |
| 11 | 60 | F | LE | Dro 3yrs | Cateract extraction – 3yrs | 6/24 | N | A | N | III,IV,VI | NAD | 9 | 90 | -Ve | NAD | NS | 2 months-LF |
| 12 | 11 | M | LE | Dip- 5D | Fever/ATT – irregular | 6/6 | N | A | N | VI | NAD | 20 | 69 | +Ve | ?PT | NS | Ref.toTB Hospital-LF |
| 13 | 64 | M | LE | H/A – Dro Def Movt. 10 D | HT | 6/18 | IMC | A | Gr-II HTR | III | NAD | 14 | 91 | -Ve | NAD | NS | 3 months-FR |
| 14 | 20 | M | LE | Dip. H/A – 2Wks | Inj. Cricket Ball. | 6/6 | N | A | N | VI | NAD | 5 | 78 | -Ve | NAD | NS | 1 months-FR |
| 15 | 52 | M | RE | Dip. H/A – 2wks | Pain | 6/18 | N | N | Colobama ONH | VI | NAD | 26 | 82 | -Ve | NAD | NS | 4 months-FR |
| 16 | 31 | M | LE | Dip. HA -1week | - | 6/6 | N | A | N | VI | NAD | 22 | 79 | -Ve | NAD | NS | 2 months-PRLF |
| 17 | 27 | M | LE | Dip. Pain – 5D | - | 6/6 | N | A | N | VI | NAD | 27 | 84 | -Ve | NAD | NS | 2 months-FR |
| 18 | 31 | M | BE | Defvn Dip – 2wks | - | 6/6 | N | A | N | VI BE | NAD | 31 | 82 | -Ve | NAD | NS | 4 months-FR |
| 19 | 30 | M | LE | Dip HA -1week | HT under trt | 6/6 | N | A | Gr1-HTR | VI | NAD | 12 | 90 | -Ve | NAD | NS | 3 months-FR |
| 20 | 5 | F | RE | Defvn 3yrs | Marcus Gunn +ve | 6/18 | N | A | N | Cong -III | NAD | - | - | - | NAD | NS | NR |
| 21 | 28 | M | LE | Dip. HA -1 yrs | Trauma | 6/6 | N | A | N | IV | NAD | 6 | 81 | -Ve | NAD | - | 1 moth –LF |
| 22 | 44 | M | RE | Dip. -10D | - | 6/12 | N | A | N | VI | NAD | 31 | 79 | -Ve | NAD | - | 3 Month - FR |
| 23 | 35 | F | LE | Drop. 3weeks | Trauma | 6/6 | N | NA | N | III | NAD | 9 | 95 | -Ve | Sphenoditis Mastoidits | NS | 4Month-FR |
| 24 | 53 | M | LE | Dip.pain 2 days | HT Undert trt | 6/9 | N | A | Gr1-HTR | VI | NAD | 10 | 106 | HS | NAD | NS | 1 ½ Months-PR LF |
| 25 | 49 | M | RE | Dip 1 months pain 15D | Ht under trt ? Pontine hge | 6/12 | N | A | Gr1-HTR | VI | NAD | 8 | 95 | -Ve | NAD | - | LF |
| 26 | 20 | M | LE | Dip. Pain -3D | - | 6/6 | N | A | N | III | NAD | 12 | 96 | -Ve | NAD | NS | 2 months-PR |
| 27 | 55 | F | LE | Dip. 1 week | - | 6/60 | IMC | A | N | III | NAD | 14 | 108 | -Ve | NAD | NS | 3 months-FR |
| 28 | 28 | M | RE | Dip. 3 months | RTA with inter hemispheric bleed | 6/6 | N | A | N | IV | NAD | 8 | 91 | -Ve | # R-panetal | - | LF |
| 29 | 35 | M | RE | Dip. 1 month | Inj with LOC. | 6/18 | N | A | N | VI | NAD | - | - | - | # L Temporal | # L- Temporal & concussion | 1 Months-NR |
| 30 | 37 | F | RE | Dip. HA -3 days | - | 6/9 | N | A | N | VI | NAD | 5 | 94 | -Ve | NAD | NS | 3Month-FR |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 31 | 24 | M | BE | Loss of vision 1year | Known case of Parietooccipital astrocytoma | No PL | RE – N LE – Total Leucoma | NA | RE-SOA | Re – VI Le-III, IV,VI | ↓ tone power sensation R side | - | - | - | - | - | Referred to neurology – LF |
| 32 | 42 | F | LE | Dip drop 3 days | - | 6/6 | N | A | N | III | N | 7 | 74 | -ve | NAD | NS | 3 months -FR |
| 33 | 28 | F | RE | Pain & Dip 3 weeks | Exanthematous Feve + | 6/35 | Iridocyclitis | NA | N | III,VI | N | 11 | 97 | -ve | NAD | - | 3 months -FR |
| 34 | 35 | M | RE | Dip.Drop.H/A 1week | - | 6/6 p | N | NA | N | III | N | 10 | 89 | -ve | NAD | NS | 3 months –FR |
| 35 | 11 | M | RE | Pain /Drop 2 yrs | H/O trauma –RTA 2 Years ago | 6/18 | N | NA | N | III | N | 11 | - | -ve | NAD | NS | Advised occlusion LE –LF |
| 36 | 55 | F | LE | Dip. Def movt 20 day | Diabetes & HT | 6/12 | IMC | A | NPDR | III,IV,VI | N | 14 | 345 | -ve | NAD | NS | 4 months –FR |
| 37 | 60 | M | LE | Pain drop 10 day | - | 6/9 | N | A | N | III | N | 8 | 101 | -ve | NAD | NS | 3 months –FR |
| 38 | 14 | M | BE | H/A 3 day | Vomiting. altered sensorium | 6/6 | N | A | Hyperemic disc | VI BE | N | - | - | - | - | - | Ref. To neurology emergency LF |
| 39 | 25 | F | LE | Dip. 2 day | - | 6/9 | N | A | N | VI | N | 16 | 78 | -ve | NAD | NS | 3 months –FR |
| 40 | 4 | F | LE | Dip .drop.4 day | Fever similar com 6 months ago | 6/9 | N | NA | N | III | N | 12 | - | -ve | NAD | NS | 2 months –PR LF |
| 41 | 34 | F | RE | Dip.partial drop 10 day | - | 6/6 | N | A | N | III | N | 8 | 74 | -ve | NAD | NS | 4 months -FR |
| 42 | 44 | F | RE | Dip –10 day | Diabetes – 10 yrs | 6/6 | N | A | NPDR | IV | N | 18 | 279 | -ve | NAD | NS | 4 months -FR |
| 43 | 36 | F | LE | Pain Dip H/A –2 day | - | 6/18 | N | A | N | VI | N | 12 | 88 | -ve | NAD | NS | 3 months -FR |
| 44 | 59 | M | RE | Dip 10 day | HT on Examination | 6/6 | N | A | N | VI | N | 14 | 92 | -ve | NAD | NS | 2 months -PR LF |
| 45 | 41 | F | LE | Dip. 15 day | - | 6/6 | N | A | N | VI | N | 8 | 84 | -ve | NAD | NS | 4 months -FR |
| 46 | 59 | F | RE | Dip 1 week | HT on Examination | 6/6 | N | A | N | VI | N | 16 | 270 | -ve | NAD | NS | 3 months -FR |
| 47 | 30 | M | RE | Dip 15 day | - | 6/6 | N | A | N | VI | N | 12 | 78 | -ve | NAD | NS | 4 months -FR |
| 48 | 21 | F | LE | Dip 2 week | - | 6/6 | N | A | N | VI | N | 10 | 88 | -ve | NAD | NS | 3 months -FR |
| 49 | 48 | M | LE | Dip.1 months | H/O RTA | 6/6 | N | A | N | VI | N | 8 | 92 | -ve | # R Panetal | #R Parietal | 4 months -FR |
| 50 | 40 | M | RE | Dip.drop 1 week | - | 6/6 | N | A | N | III | N | 14 | 84 | -ve | NAD | NS | 2 months -NR |
| 51 | 18 | F | BE | H/A.Pain def.movt 10 day | Suspect demyeli | 6/6 | N | A | N | VI, BE | N | 20 | 76 | -ve | NAD | - | LF |
| 52 | 47 | F | LE | Drop15 day | - | 6/18 | N | NA | N | III | N | 16 | 85 | -ve | NAD | NS | 3 months -FR |
| 53 | 65 | M | RE | Drop 1 week | Diabetes | 6/6 | N | A | N | III | N | 7 | 180 | -ve | NAD | NS | 3 months -FR |
| 54 | 28 | M | RE | Dip 1 week | Vomiting | 6/6 | N | A | N | VI | N | 9 | 92 | -ve | NAD | NS | 1 month –FR2 months later III N-FR |
| 55 | 40 | F | LE | Dip.H/A Pain –2 months | - | 6/12 | N | A | N | VI | N | 11 | 87 | -ve | NAD | NS | 2 months -FR |
| 56 | 55 | M | RE | Dip.Partial drop 1 months | - | 6/60 | IMC | NA | N | III.IV,VI & VI | N | 28 | 106 | -ve | SOF – Penosteal Thick | - | 3 months -FR |
| 57 | 18 | F | LE | Drop-from childhood | - | 1/60 | N | NA | N | III | N | 15 | 75 | -ve | NAD | NS | NR- Congenital |
| 58 | 25 | F | LE | Dip.Pain-2 week | - | 6/9 | N | A | N | VI | N | 17 | 98 | -ve | NAD | NS | 2 months -FR |
| 59 | 42 | F | LE | H/A drop 1 months | - | 6/9 | N | NA | N | III | N | 15 | 124 | -ve | NAD | NS | 3 months -FR |
| 60 | 33 | M | RE | Dip 2 months | H/O RTA | 6/6 | N | A | N | IV | N | 7 | 80 | -ve | - | Contusion R frontal Labe | 2 months -FR |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 61 | 50 | F | LF | Dip. one month | H/O RTA With LOC ans bleeding thro mouth and nose | 6/6 | N | A | N | VI | N | 6 | 78 | -Ve | #R ParIetal bone | Contusion R Parietal regIon | 2Months NR LF |
| 62 | 34 | M | RE | Dip. 1 week H/A 2 weeks | Fever 1 week | 6/6 | N | A | Bilateral Papiledem a | VI | N | 8 | 86 | -Ve | Erosion of Post Clinoid | - | LF |
| 63 | 29 | M | RE | Pain. Dip 20 days | - | 6/9 | N | A | N | VI | N | 12 | 74 | -Ve | NAD | NS | |
| 64 | 40 | F | LE | H/A dip.pain 1 month | - | 6/12 | N | A | N | VI | N | 20 | 80 | -Ve | NAD | NS | |
| 65 | 50 | M | RE | Dip. 2 weeks | HT of Examination | 6/9 | N | A | N | VI | N | 18 | 76 | -Ve | NAD | NS | |
| 66 | 23 | M | RE | Dip. Pain 1 month | - | 6/9 | N | A | N | VI | N | 12 | 84 | -Ve | NAD | NS | |
| 67 | 67 | M | RE | Dip. One month | HT | 6/60 | IMC | A | N | VI | N | 18 | 98 | -Ve | NAD | NS | |
| 68 | 49 | M | LE | Dip. 1 month | - | 6/12 | N | A | N | III | N | 20 | 76 | -Ve | NAD | NS | |
| 69 | 45 | F | RE | Dip. Pain 3 days | Diabetes | 6/36 | IMC | A | Temporal pallor | III.IV.VI | N | 14 | 312 | -Ve | NAD | NS | |
| 70 | 69 | M | RE | Drop.Dip. 15 days | - | 6/18 | IMC | A | N | III | N | 11 | 84 | -Ve | NAD | NS | |
| 71 | 18 | F | LE | Dip. Pain 1week | - | 6/6 | N | A | N | VI | N | 14 | 83 | -Ve | NAD | NS | |
| 72 | 13 | M | BE | Def.vn 10 days | Fever alt sensonum 10 days | 6/18 | N | Stug gil | Temporal pallor | VI | Planter I | 13 | 80 | -Ve | Erosion of Clinoid | Hypodense mass in Sellar regin | 3 months FR |
| 73 | 18 | F | LE | H/A Left ear ache + dischage 1 month | CSOM left | 6/6 | N | A | N | VI | N | 17 | 74 | -Ve | Mastoditias d | - | 4 Months FR |
| 74 | 10 | F | RE | H/A 1 week | H/O Epilepsy once | 6/6 | N | A | N | VI | N | 11 | 76 | -Ve | NAD | NS | 3 months FR |
| 75 | 26 | F | RE | Dip. 1 month | Mass R nostril irradiation Given | 6/6 | N | A | N | VI | N | 5 | 85 | -Ve | - | - | 3 months FR |
| 76 | 64 | M | LE | Drop.RE 1 week | Diabetes | 6/60 | IMC | A | N | III | N | 5 | 145 | -Ve | NAD | NS | 3 months FR |
| 77 | 57 | M | LE | Dip.1 ½ months | Diabetes | 6/24 | IMC | A | N | VI | N | | 180 | - | NAD | NS | 10 Days LF NR |
| 78 | 21 | F | RE | Dip.partial drop 2 months | Hypothyroid | 6/9 | N | A | N | III | N | 8 | 90 | - | NAD | NS | 4 Months FR |
| 79 | 25 | F | RE | Dip.Def.movt. 10 days | - | 6/12 | N | A | N | VI | N | 12 | 70 | -Ve | NAD | NS | 4 Months FR |
| 80 | 30 | M | RE | Def. Movt.10 days | Def.VnRE since childhood | 1/60 | N | A | Highmyop ia | VI | N | 7 | 82 | -Ve | NAD | NS | 3 Months FR |
| 81 | 47 | F | LE | Def.movt.drop. 4 moths | - | 3/60 | IMC | Stug gil | N | III.IV.VI | N | 25 | 79 | -Ve | Sinus maxillary growth | - | Taken by neurology dept |
| 82 | 21 | M | RE | Dip. 10 days | - | 6/6 | N | A | N | IV | N | 6 | 92 | -Ve | NAD | - | 3 Months FR |
| 83 | 30 | M | RE | H/A 5 months Dip. 1 months | - | 6/6 | N | A | N | VI | N | 8 | 88 | -Ve | NAD | - | 3 Months FR |
| 84 | 45 | F | RE | Dip. 1 ½ moths | - | 6/6 | N | A | Temporal pallor | VI | N | 6 | 66 | -Ve | NAD | - | ? Nasopharyngal TR Referred to ENT |
| 85 | 15 | F | RE | Def. Vn pain 1 week | Case of JNA ref. from ENT | PL | N | A | POA | III & III | N | - | - | - | - | - | 3 Months FR |
| 86 | 21 | M | RE | Dip. 2 months | H/o injury ref.from ENT | 6/6 | N | A | N | IV | N | 9 | 81 | -Ve | NAD | - | 3 Months FR |
| 87 | 23 | F | LE | Drop.2weeks | - | 6/6 | N | A | N | VI | N | 8 | 78 | -Ve | NAD | - | 2 Months FR |
| 88 | 58 | M | RE | Dip.4 days | Diabetes | 6/12 | N | A | N | VI | N | 20 | 194 | -Ve | NAD | - | 3 ½ months FR |
| 89 | 52 | M | RE | Drop.pain H/A 1 week | HT | 6/12 | IMC | NA | GRII HT Changes | III | N | 11 | 87 | -Ve | NAD | - | 3 Months FR |
| 90 | 19 | M | LE | Partial Drop Det. Movt 3 months | H/O Accident 3 months with LOC | NO PL | N | NA | POA | III | N | 6 | 78 | - | - | - | 3 months FR NR V/A |
| 91 | 58 | F | RE | Def Mov.10days | Nil | NOP L | N | RAPD | Temporal Pallor | III.IV-III | NAD | 16 | 108 | -ve | NAD | - | Ref. to Diabetology LF |
| 92 | 29 | M | RE | Dip.drop.pain 3days | H/O ink RE with stick | 6/24 | N | Sluggish | Macular Edema | III | NAD | 6 | 86 | - | NAD | - | 3 months FR |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 93 | 19 | M | Re | Dip.drop.pain 1month | - | 6/6 | N | Sluggish | n | III | - | 8 | 90 | - | NAD | NS | 2 months FR |
| 94 | 35 | M | LE | Dip.2 weeks | - | 6/6 | N | A | Myopic | VI | NAD | 6 | 86 | -ve | NAD | NS | 3 months FR |
| 95 | 54 | M | LE | Dip Def.Vn.10 days | HT | 4/70 | IMC | A | GRII HT Change | VI | NAD | 10 | 92 | - | NAD | NS | 3 months FR |
| 96 | 20 | M | BE | Dip.21/2 weeks | - | 6/6 | N | A | N | VI | NAD | 17 | 86 | -ve | NAD | NS | 1 month pr |
| 97 | 24 | F | RE | Dip 2 weeks | - | 6/6 | N | A | N | VI | NAD | 13 | 79 | - | NAD | NS | 3 months FR |
| 98 | 65 | M | LE | Dip-3 Days | Diabetes | 6/18 | IMC | A | N | VI | NAD | 15 | 192 | -ve | NAD | NS | 3 months FR |
| 99 | 60 | F | LE | Partial drop. Defmovtpain dip 2weeks | - | 6/18 | IMC | A | N | III.IV & VI | NAD | 13 | 78 | - | SOF –2 thickened periostium | NS | 3 months FR |
| 100 | 39 | M | RE | Dip.Def.Vin.2Weeks | - | 6/6 | N | A | N | IV | NAD | 8 | 86 | -ve | NAD | NS | 3 months FR |
| 101 | 65 | M | RE | Pain H/A Dip.15 days | Diabetes | 5/60 | IMCA | A | N | VI | NAD | 12 | 214 | - | NAD | NS | 3 months FR |
| 102 | 32 | M | RE | Dip.31/2 months | H/O Trauma | 6/24 | N | A | N | VI | NAD | 11 | 82 | -ve | - | III Def. hypodense area R occiput | 30 days PR LF |
| 103 | 23 | M | LE | H/A Dip.15 days | - | 6/6 | N | A | N | VI | NAD | 8 | 88 | -ve | NAD | NS | 3 months FR |
| 104 | 42 | F | RE | Dip.3 years | Diabetes | 6/18 | IMC | A | N | VI | NAD | 10 | 218 | -ve | NAD | NS | 3 months FR |
| 105 | 27 | M | RE | Dip.5 years | H/O Trauma | 6/6 | N | NA | N | III | NAD | 6 | 83 | -ve | Old # Temp bone | # Temp. bone | NR |
| 106 | 70 | M | LE | Dip Partial drop 5 days | - | 6/6 | N | A | N | III | NAD | 16 | 93 | -ve | NAD | NS | 3 months FR |
| 107 | 28 | F | RE | Drop. Pain 10 days | - | 6/6 | N | A | N | III | NAD | 13 | 76 | -ve | NAD | NS | 3 months FR |
| 108 | 2 | FC | LE | Squinting LE 10days | Fever 20 days | - | N | A | N | III | NAD | 8 | - | -ve | NAD | NS | 3 months FR |
| 109 | 19 | F | BE | H/A dip. Pain 10 days | Fever neck rigidify | 6/6 | N | A | Papilloedema | VI | NAD | 22 | 79 | +ve | NAD | NS | 2 months PR on ATT |
| 110 | 40 | F | LE | Dip.10 days | - | 6/6 | N | A | N | VI | NAD | 14 | 83 | -ve | NAD | NS | 3 months FR |
| 111 | 65 | M | LE | Dip.1 months | HT | 5/60 | IMC | A | NO View | VI | NAD | 9 | 94 | -ve | NAD | NS | 3 months FR |
| 112 | 63 | M | LE | Dip. pain 1 week | Diabetes | 6/9 | N | A | N | VI | NAD | 18 | 270 | - | NAD | NS | 3 months FR |
| 113 | 70 | M | LE | Dip.10 days | HT | 6/24 | IMC | A | GRII Change | VII | NAD | 7 | 82 | - | NAD | NS | 3 months FR |
| 114 | 60 | F | RE | Complete drop 11/2months | - | 6/24 | IMC | NA | N | III,IV VI | NAD | 6 | 96 | -ve | NAD | Basal Meningoma | NR Ref to Neuro |
| 115 | 10 | F | LE | Def.Vn.Pairtical drop crop 2 months | Post TB meningitis | CF CF | N | NA | POA | III,VI | NAD | - | - | +-ve | - | - | PR 4 months on ATT |
| 116 | 34 | M | BE | Dip.4 days | - | 6/6 | N | A | Papilloedema | VI | - | 9 | 112 | +ve | NAD | NS | PR 4 months on ATT |
| 117 | 62 | F | LE | Dip. Pain 15 days | - | 6/12 | IMC | A | N | VI | NAD | 8 | 82 | -ve | NAD | NS | 3 months FR |
| 118 | 6 | F | LE | Squinting LE with face turn | - | 6/6 | N | A | N | VI | NAD | 12 | - | +ve | NAD | - | Ref. paediatric neurology LF |
| 119 | 35 | F | LE | Drop pain 1 month | | 6/6 | N | A | N | III IV Vi | NAD | 11 | 100 | -ve | NAD | NS | 3 months PR |
| 120 | 35 | M | RE | Drop 15 days | | 6/12 | N | A | N | IV | NAD | 33 | 82 | -ve | NAD | NS | 3 months PR |
| 121 | 14 | F | RE | Dip.1 month | - | 6/9 | N | A | Temporal pallor | VI | NAD | 21 | - | -VE | NAD | NS | 3 moths FR |
| 122 | 24 | F | LE | Dip. 1 ½ months | - | 6/6 | N | A | N | VI | NAD | 18 | 86 | -VE | NAD | NS | 1 month FR |
| 123 | 50 | F | BE | Drop.Def.mov.since childhood | - | 6/60 | IMC | A | Temporal pallor | III, IV, Vi | NAD | - | - | - | - | NS | Congenital – NR |
| 124 | 46 | F | LE | Pain Dip. 1 week | Nil | 6/60 | IMC | A | N | VI | NAD | 24 | 96 | -VE | NAD | NS | 3 months FR |
| 125 | 18 | M | RE | Pain Dip. 1 day | - | 6/60 | N | A | N | VI | NAD | 38 | 74 | -VE | NAD | NS | 3 months FR |
| 126 | 70 | M | RE | Drop. 10 days | Diabetes | 3/60 | IMC | NA | BDR | III | NAD | 32 | 182 | -VE | NAD | NS | 3 months FR |
| 127 | 49 | F | LE | Dip. 4 days | L VII palsy 10 years ago | 6/60 | IMC | A | N | VI | NAD | 20 | 150 | -VE | NAD | NS | 3 months FR |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 128 | 25 | M | RE | Def.Mov.partial drop. 1 month | H/O RTA with LOC 1 month go | 6/12 | N | Sluggish | N | III | NAD | 16 | 96 | -VE | # parietal bone | # parietal bone | 3 months PR |
| 129 | 60 | M | RE | H/A drop 1 month | Diabetes | 1/60 | IMC | NA | N | III | NAD | 22 | 185 | -VE | NAD | NS | 3 months FR |
| 130 | 52 | M | RE | Drop. H/A 1 week | Diabetes, IHD 1 months | 6/18 | IMC | A | N | III | NAD | 7 | 185 | - | NAD | NS | 3 months FR |
| 131 | 27 | M | LE | Dip. 40 days | -- | 6/6 | N | A | N | VI | NAD | 7 | 82 | -VE | NAD | NS | 3 months FR |
| 132 | 22 | M | RE | Dip. 3 weeks | - | 6/9 | N | A | N | VI | NAD | 12 | 78 | -VE | NAD | NS | 2 months PR |
| 133 | 63 | M | RE | Drop. 1 month | Diabetes | 5/60 | Aphakia | A | N | III | NAD | 10 | 178 | -VE | NAD | NS | 3 months FR |
| 134 | 50 | F | RE | Partial drop dip. 15 days | HT | 6/60 | IMC | A | GR1HT Changes | III | NAD | 60 | 140 | -VE | NAD | NS | 4 months FR |
| 135 | 28 | M | RE | Dip. 10 days | - | 6/18 | N | A | N | VI | NAD | 28 | 88 | -VE | NAD | NS | 3 months FR |
| 136 | 48 | M | RE | Dip.pain H/A 2 days | - | 6/6 | N | A | N | VI | NAD | 24 | 190 | -VE | NAD | NS | 3 months FR |
| 137 | 55 | M | LE | Dip. 1 week | - | 6/12 | IMC | A | N | VI | NAD | 11 | 126 | - | NAD | NS | 3 months FR |
| 138 | 27 | F | RE | Dip.pain H/A 10 days | - | 6/6 | N | A | N | IV | NAD | 14 | 90 | -VE | NAD | NS | 2 months FR |
| 139 | 40 | M | LE | Def.vn 3 months | - | 6/18 | N | NA | N | III | NAD | 18 | 90 | -VE | NAD | NS | 2 months PR LF |
| 140 | 68 | F | LE | Def.vn 1 week | HT | CF CF | MC | A | No View | VI | NAD | 7 | 94 | -VE | NAD | NS | 3 months FR |
| 141 | 7 | M | RE | H/A Dip. 1 week | Fever | NC | N | A | N | VI | NAD | 24 | - | -VE | NAD | NS | 3 months FR |
| 142 | 42 | M | LE | Dip. 1 month | H/O Trauma left side of face. | 6/6 | N | A | N | VI | NAD | 9 | 86 | -VE | NAD | NS | 3 months FR |
| 143 | 45 | M | LE | Drop. 1 week | H/O Trauma | 6/6 | N | NA | N | III | NAD | 26 | 140 | -VE | NAD | NS | 2 months PR |
| 144 | 50 | M | RE | Dip. pain 10 days | Diabetes | 5/60 | IMC | A | N | VI | NAD | 21 | 168 | -VE | NAD | NS | 3 months FR |
| 145 | 60 | M | RE | Dip. 10 days | Diabetes | 6/6 | N | A | N | III | NAD | 28 | 80 | -VE | NAD | NS | 3 months FR |
| 146 | 30 | F | RE | Dip. 1 ½ months | - | 6/6 | N | A | N | III | NAD | 15 | 70 | -VE | NAD | NS | 1 ½ months PR |
| 147 | 38 | F | LE | Dip. H/A 15 days | - | 6/9 | N | A | N | VI | NAD | 25 | 100 | +VE | No PT | NS | 3 months FR |
| 148 | 40 | F | LE | H/A dip. pain partial drop 15 days | Diabetes | 8/60 | IMC | NA | N | III,IV,VI | NAD | 40 | 208 | +VE | NAD | NS | 3 months FR |
| 149 | 10 | F | BE | Def. mov | - | HM | N | A | Pallor | III,IV,VI | - | - | - | - | - | NS | NR – congenital |
| 150 | 51 | M | RE | Pain 10 days | H/O trauma | N | NA | N | N | III | NAD | 28 | 70 | -VE | NAD | NS | 3 months PR |
| 151 | 23 | F | BE | Loss OF Visio 1 month | Fever – Encephalitis | No peal | N | NA | Bilateral Papiloedema | VI | NAD | 40 | 75 | -VE | NAD | - | NR |
| 152 | 45 | M | RE | Dip. 10 days | - | 6/24 | IMC | A | N | VI | NAD | 17 | 100 | -VE | NAD | NS | 3 moths FR |
| 153 | 30 | F | RE | Dip. 1 week | - | 6/9 | N | A | N | VI | NAD | 13 | 75 | -VE | NAD | NS | 3 moths FR |
| 154 | 40 | M | BE | Dip. H/A 15 days | - | 6/18 | N | A | N | VI | NAD | 7 | 185 | -VE | NAD | NS | Ref. To Diabetology |
| 155 | 30 | F | RE | Dip. Face turn 10 days | - | 6/9 | N | A | N | VI | NAD | 9 | 74 | -VE | NAD | NS | 3 moths FR |
| 156 | 60 | M | LE | Dip. 15 days | Diabetes | 6/9 | N | A | N | VI | NAD | 12 | 180 | - | NAD | NS | 3 moths FR |
| 157 | 1 ½ | M | RE | Squinting RE dip. 2 days | H/O Trauma R site of face | NC | N | A | N | VI | NAD | - | - | -VE | NAD | NS | 2 moths FR |
| 158 | 28 | M | RE | Head tilt dip 2 years | H/O Head inj.2 years ago | 6/6 | N | A | N | IV | NAD | - | - | - | - | NS | NR |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 159 | 62 | M | RE | Dip. Pain 7 days | Diabetes | 6/12 | N | A | NPDR | VI | NAD | 28 | 248 | - | NAD | NS | 3 moths FR |
| 160 | 29 | M | RE | Drop. 2 weeks | - | 6/6 | N | A | N | III | NAD | 28 | 90 | -VE | NAD | NS | 2 moths PR LF |
| 161 | 45 | M | RE | Partial drop. Dip. 30 days | H/O fall from bike | 6/6 | N | Sluggish | N | III | NAD | 7 | 86 | -VE | NAD | NS | 3 moths PR |
| 162 | 17 | F | BE | Dip. 4 days | Fever with neck stiffness 4 days | 6/6 | N | A | Hyperemia | VI | NAD | 14 | 69 | -VE | NAD | - | Sent neurology |
| 163 | 41 | M | RE | H/A Dip.1week | - | 6/6 | N | A | N | VI | NAD | 24 | 96 | -VE | NAD | NS | 1 months FR |
| 164 | 14 | F | LE | Dip 10 days | - | 6/6 | N | A | N | VI | NAD | 24 | 72 | -VE | NAD | NS | 3 moths FR |
| 165 | 45 | F | LE | Drop. 1 week | Diabetes | 6/6 | N | A | N | III | NAD | 18 | 212 | -VE | NAD | NS | 3 moths FR |
| 166 | 32 | F | RE | Drop. 1 month | H/O Trauma Over right eyebrow | 6/18 BE | N | A | N | III | N | 9 | 87 | -VE | NAD | - | Neurology LF |
| 167 | 64 | M | RE | Complete drop 10 days | - | HM | MC | A | No view | III | N | 8 | 103 | -VE | NAD | NS | 3 moths FR |
| 168 | 33 | M | LE | Dip. Pain 1 week | - | 6/9 | N | A | N | VI | N | 15 | 79 | -VE | NAD | NS | 1 moth PR LF |
| 169 | 60 | M | RE | Dip partial drop 1 week | HT | 6/18 | IMC | A | N | III | N | 6 | 98 | - | NAD | NS | 2 moths PR |
| 170 | 55 | M | RE | Dip. 1 week | HT | 6/9 | N | A | N | VI | N | 8 | 84 | -VE | NAD | NS | 3 moths FR |